Vascular Surgery

By

GEZA de TAKATS, M.D., M.S., F.A.C.S.

Clinical Professor of Surgery, University of Illinois
College of Medicine; Attending Surgeon,
Presbyterian-St. Luke's Medical Center and Research and
Educational Hospitals, Chicago, Illinois.

We need to cut passages between shafts we have already dug instead of merely digging the same old shafts deeper and deeper. — STRINGFELLOW BARR

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FOR CAROL

PREFACE

IT HAS BEEN AN OLD CUSTOM FOR THE AUTHOR TO GREET HIS READERS WITH appropriate salutations and to so whet their appetite that they would follow his discourse from beginning to finish I however must begin by stating that in these times of rapid technical advance it is almost inevitable that a monograph embodying many technical procedures be out of date in some respects even on the day of its publication This prospect is a discouraging one to any author In the present instance I have been borne up by the conviction that it is in the very areas where technical advances are most frequent that there is greatest need for a statement of fundamental principles There must remain a home base a foundation of basic facts which do not change even though their interpretation may have to be modified from time to time

The subject of vascular surgery seemed simple and was largely neglected 30 years ago At that time Allen B Kanavel gave generous support to the establishment of a clinic in vascular surgery at Northwestern University From here it was transplanted to the University of Illinois under the acting chairmanship of Eric Oldberg, who first placed this subject in the senior surgical curriculum In Warren H Cole s Department of Surgery the clinic flourished and expanded to an active cardiovascular service At St Luke s Hospital a Cardiovascular Surgical Service was established to which with the help of the late Huntington Henry our Board Chairman of great vision and foresight a cardiovascular research unit was attached

Without all these men and without the loyal and faithful cooperation of many internists notably N C Gilbert G K Fenn and George W Scupham the present volume could not have been written When the reader is invited to take a trip with us through some of the charted and often uncharted seas it must be made clear that only personal experience will be registered and that many areas in which I myself have not sailed, have not been included in this monograph There is no cardiac or cardiothoracic subject discussed unless it bears on peripheral circulation

It is precisely the dramatic impact of cardiothoracic surgery which makes it necessary to assemble information on the many facets of vascular disease requiring surgical care It need not be inevitable that pump-oxygenators cardiac bypasses and septal defects will so absorb the energies and attention of surgical staffs that many vascular problems which are still far from being solved will be relegated to the junior resident

It is the purpose of this volume to describe the experience of our group It is only through a good backward look into the development of vascular surgery in our own institution that the present advances can be understood and assessed As in any other surgical endeavors, technical skill is important and should be practiced and standardized in the experimental laboratory But boldness and manual dexterity in the human cannot be substituted for sound judgment, and sound judgment can only be acquired by knowing the natural course of the disease. The natural course of the disease does not manifest itself in the operating room or the laboratory and demands full attention from those who are caring for the total patient and not just part of him

This is not to say, however, that surgical technique has been slighted in this book Vascular grafts and endarterectomies are here to stay, will become simpler and will become part of the armamentarium of every well trained surgeon But as their limitation becomes obvious with the course of time, many patients should be spared the argument that "let us try it, we can always amputate later " Precise indications and proper timing need emphasis over and over again and this I have tried to do on the basis of what is known at this writing

Nor do I believe that sympathectomies are obsolete, that they are superseded and that they have no more place in vascular surgery, it is a commonplace experience that the optimal solution of an advanced vascular insufficiency may be resolved by a combination of sympathectomy, restoration of arterial continuity and a minor amputation

The illustrations have been prepared by Gloria Jones and Kathleen Mullen Hentges Professor A Hooker Goodwin, of our Illustration Studios, acted as their advisor and consultant Helene Coleman, Mary Van Nortwick, Dona Gilbo and Patricia Samsel toiled with the manuscript George F Knisely of St Luke's Hospital made the photographs

Edson Fairbrother Fowler, my former associate, read the galley proofs, Miss Angeline McNeill, Medical Librarian of St. Luke's Hospital, was of great help with the bibliography, and Miss Margaret Doherty prepared the index

My deep gratitude goes to the staff of W B Saunders Company, who patiently steered the fitful voyage of this cruiser through Scylla and Charybdis into what we hope is a safe harbor

GEZA DE TAKATS

Chicago, Illinois

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PART I

Fundamental Principles Affecting Vascular Surgery

Teleology is a lady without whom no biologist can live Yet he is ashamed to show himself with her in public

E VON BRUCKE, 1896

INTRODUCTION

IF THE SURGEON WISHES TO BE MORE THAN A SKILLED TECHNICIAN HE MUST understand the impact of his operations on the total function of the body. There are a number of excellent studies of applied physiology of the vascular tree Here we have especially relied on the writings of Wiggers,[1] Burton,[2] the symposia of the Ciba Foundation on visceral and peripheral circulation[3, 4] and the monograph of Barcroft and Swan [5] The chapter on blood clotting is based on a recent short monograph by the author [6] A study of these basic factors may make the vascular surgeon both more radical and more conservative It will persuade him to attack the vascular problem early with simple measures, it may also cause h m to refrain from subtotal resections of the abdominal aorta when the patient is disintegrating from a diffuse, advanced cardiovascular renal disease The material in this part has been used in a lecture delivered before the New Orleans Surgical Society in 1956 [7]

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CHAPTER 1

HEMODYNAMICS

THE CLOSED SYSTEM OF HEART, ELASTIC AND MUSCULAR ARTERIES, TERMINAL vessels and veins constitutes an apparatus in which many physical factors operate to ensure an adequate blood flow The central problem is the relation between the rate of flow of blood and the pressure that drives it This has been ingeniously analyzed in the writings of Burton [1] [2] He defined the criti cal closing pressure as a disturbed state of equilibrium at which a vessel would close actively and completely when the pressure within it falls below a certain critical value The critical closing pressure increases with increasing tension in the wall (vascular tone) and with decreasing size of the vessel

Thus vascular spasm simply means from a hemodynamic standpoint, that the critical closing pressure is higher than the available blood pressure In conditions of high vasomotor tone such as in primary neurogenic ' shock certain vascular beds may completely close with resultant irreversible damage

The level of the blood pressure thus takes on a new importance in modifying the effect of vasomotor tone on circulation For instance, under sympa thetic stimulation the flow in the rabbit s ear is reduced to one half when the arterial pressure is 100 mm Hg, but to one fifth when the pressure is lowered to 60 mm Hg In the arm of a patient who had a critical closing pressure of 70 mm. Hg, lowering the arterial pressure by 20 mm Hg resulted in an 80 per cent decrease of flow [3]

The significance of blood pressure with maintained or increased vasomotor tone for the peripheral circulation is obvious When hypotension is induced during operations it should be accompanied by vasomotor relaxa tion Thus hypotensive anesthesia produced by ganglionic depressants is preferable to that induced by bleeding.

Hypothermia combined with Thorazine or Arfonad is safer than if vasoconstriction is allowed to prevail as a response to chilling the body This principle finds daily application in controlled hypotension an important technique in vascular surgery

The separate and combined mechanical functions of the four kinds of tissue in the wall of blood vessels, namely endothelium, elastic fiber smooth muscle and collagen fibers, have special bearing on the fate and function of vascular grafts The tensile strength and the breaking load of aortic hemografts in the experimental animal are quite satisfactory [4] Thanks to the high tensile strength of the scar tissue the anastomoses compare favorably in

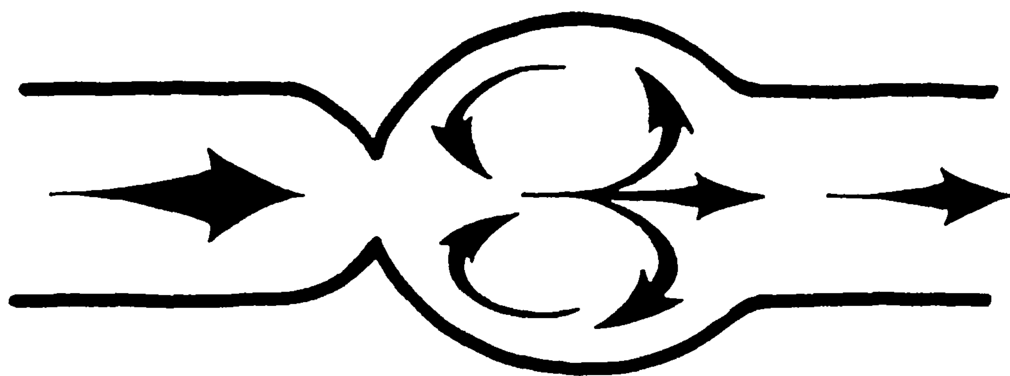


FIG 1 The effect of arterial stenosis on turbulence and reverse flow The rapidly ejected stream strikes the more slowly flowing distal stream High kinetic energy is transformed into high potential energy This is based on an injection of methylene blue (Redrawn from E Holman New Concepts in Surgery of the Vascular System Charles C Thomas, Springfield, Ill, 1955)

strength with the host aorta However, when the grafts are visualized under pressures varying from 250 to 35 mm Hg, the diameter of a normal dog's aorta will vary from 10 to 7 mm, whereas the aort c graft acts as a rigid tube and does not dilate under high pressure Thus, the end result of any vascular graft will be a more or less rigid tube caused by gradual loss of elastic fibers and by the scar produced by the host [4] Since the vessel wall decreases in elasticity with age and since the vessel transplant, whether a homologous vessel or a plastic substance, will become fibrotic in time, the effort to supply a distensible, elastic transplant seems—at present, at least—desirable, but no patient needs a more elastic graft than his own vascular tree nor is it possible to maintain such elasticity after the graft is in place

The hemodynamic effects of short, rigid conduits have certainly been satisfactory in the aortic transplants of grafts in children [5] How a long, rigid tube consisting of a transplanted artery, an autogenous vein or a synthetic substance will fare still awaits long-term follow-up studies

The direct response of an arterial or arteriolar wall to a rise in pressure is contraction Byrom[6] in a recent experimental study on hypertensive encephalopathy revived the long-forgotten teachings of Bayliss, stating that tension within the artery is the normal and direct stimulus to contraction Thus, any measure that will lower excessive tension, such as exists in the malignant phase of hypertension, should diminish vasospasm, hemorrhage and edema This is the most reasonable explanation of the improvement in eyegrounds, in encephalopathy and in focal arteriolar necrosis when blood pressure is lowered, whether this is accomplished by venesection, by diet, by hypotensive drugs or by sympathectomy

The effects of arterial stenosis have been ingeniously studied by Emile Holman,[7] who showed that "structural fatigue" will develop in segments distal to the narrowing and that the poststenotic dilatation is not due to sympathetic paralysis or compression of the vasa vasorum but can be produced in an inert rubber tube if blood is pumped through it for a sufficient length of time The turbulence of the blood and the transformation of high kinetic energy into lateral pressure exert a continuous stress on the arterial

wall (fig 1) The clinical implications of these experiments explain the aneurysms seen in subaortic stenosis, pulmonary valvular stenosis coarctations of the aorta and beyond a cervical rib (fig 2) I have repeatedly seen a stenosis at Hunter s canal proximal to popliteal aneurysms

In many older textbooks the law of Poiseuille is frequently invoked which states that the rate of flow is related to the square root of the diameter of the rigid tube As Burton[1] recently stated Poiseuille s law applies only when the flow is streamlined not turbulent when the fluid is simple and incompressible and when the tubes are nondistensible The last two of these three conditions do not prevail in vascular beds and hence Poiseuille s law does not apply to active circulation

More than a century ago a French mathematician and astrophysicist Pierre Simon Laplace announced a very simple physical law when he stated

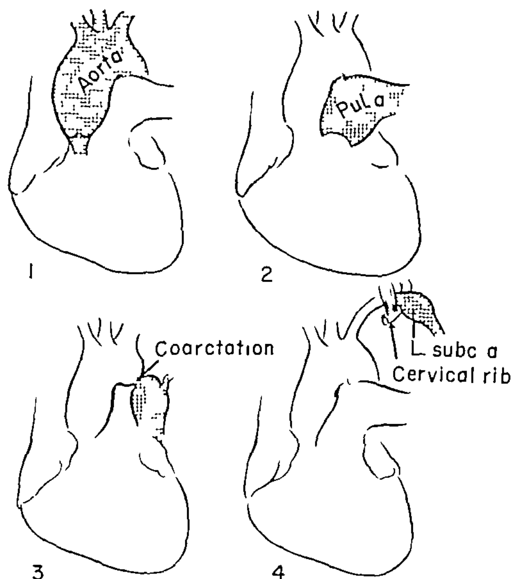


FIG 2. Poststenotic dilatation in (1) subaortic stenosis (2) pulmonary valvular stenosis (3) beyond coarctation of the aorta and (4) distal to a cervical rib The degree of dilatation is dependent on the height of pressure and the velocity of flow thus it is maximal beyond a subaortic stenosis and minimal beyond a cervical rib (E. Holman New Concepts in Surgery of the Vascular System. Charles C Thomas, Springfield Ill., 1955)

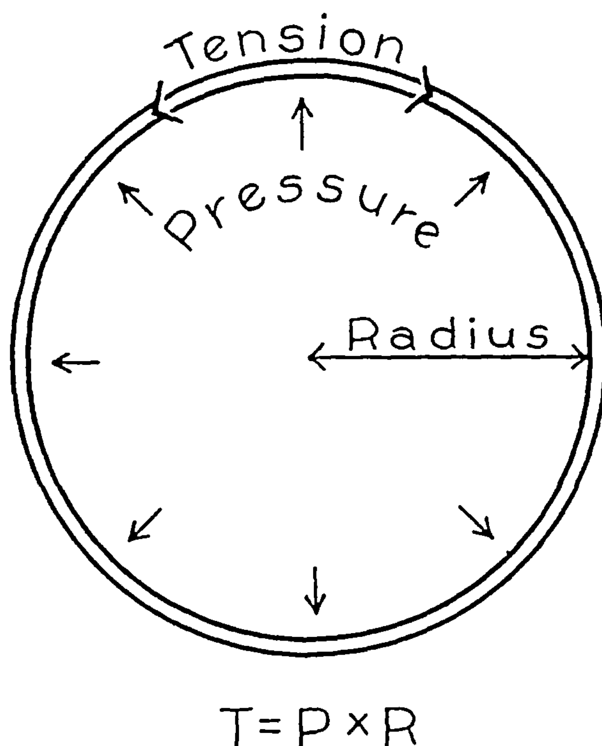


FIG 3 The Law of Laplace *Tension* of the wall equals *Pressure* multiplied by *Radius* When the pressure within a small blood vessel falls below a certain value (critical closing pressure), the vessel closes actively and completely The critical closing pressure increases with increasing tension in the wall (vasomotor tone) and with decreasing size of the vessel (Burton, A C . Law of Physics and Flow in Blood Vessels Visceral Circulation A Ciba Symposium, Little, Brown and Co , Boston, 1953)

that the tension of a cylindrical wall and the pressure within it are in equilibrium Thus, *Tension* equals *Pressure* times *Radius* (fig 3) This principle was recently used to explain the hemodynamics prevailing in aneurysms by Pirani and myself.[8]

Blood flow, velocity of flow and blood pressure have been loosely correlated in many clinical tests A critical summary of methods available for the measurement of human peripheral blood flow has been recently offered by Burton [9] We shall return to these tests in part II, Methods of Diagnosis

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CHAPTER 2

VASCULAR SHUNTS

THE CIRCULATORY SYSTEM NORMALLY FUNCTIONS AS A UNIT ANY CHANGE IN circulation in one part will have an effect, no matter how small, on the total circulation, whether somatic or splanchnic [1] This principle was enunciated by Rein[2] and stressed again by DeBakey, Burch and Ochsner [3] It is the object of functional anatomy to demonstrate the mechanisms by which such shifts of blood flow can be accomplished The vascular system has to insure two separate functions the circulation of blood as a whole and the provision of special circulatory conditions in an organ whose function needs intermittent fluctuations of blood supply The role of the vasomotor apparatus in shifting blood has been well established and will be the subject of another chapter There is, however, a structural basis for an important group of short-circuiting mechanisms whose function is to regulate the blood supply to organs or parts of the vascular tree

These mechanisms include (1) arteriovenous anastomoses (fig 4), (2) other arterial mechanisms, including special muscular ridges and sphincters which arrest blood flow at strategic points (fig 5), and (3) possible preferential channels in the capillary bed, as suggested by Chambers and Zweifach,[4] to conduct blood between arteriole or metarteriole to venule (fig 6)

Arteriovenous Anastomoses

The locations of the *arteriovenous anastomoses*, which have a very rich nerve supply (Masson[5]), are enumerated in the following summary of J D Boyd [1]

(1) Dermis, tongue, nasal mucosa
(2) Intestinal tract villi of gut, stomach
(3) Glands submandibular, thyroid
(4) Liver (hepatic artery to portal vein)
(5) Kidney
(6) Lungs (pulmonary artery to vein, bronchial artery to pulmonary artery)
(7) Genital system erectile mechanism, ovary, uterine mucosa
(8) Joints
(9) Special shunts glomus coccygeum and glomeruli caudales, carotid body and homologous structures
(10) Intervillous or labyrinthine space of the placenta
(11) Cardiac muscle, visceral muscle

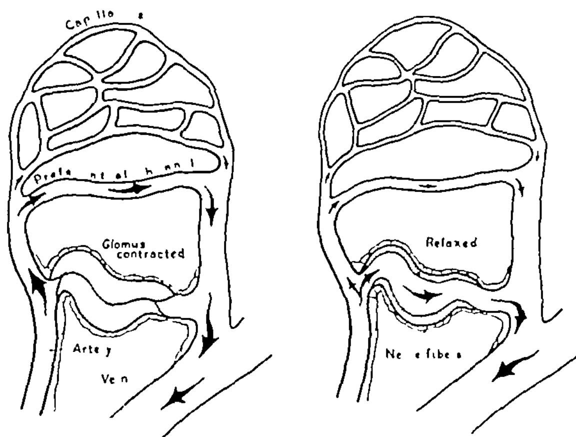


FIG. 4 Diagram of a normal glomus in a contracted and relaxed state When the shunt is closed, the arterial blood passes to the terminal vessels, but even here preferential channels may operate. When the shunt is open, the terminal vascular bed receives only a part of the arterial blood. The venous blood is arterialized. Such a condition exists shortly after sympathectomy and in the vasodilator phase of cold injury (Modified from Masson, P Les Glomus Neurovasculaires. Hermann et Cie Paris, 1937)

This is an extensive list of proven or alleged arteriovenous anastomoses Their absence in striated muscle is to be noted with surprise since as will be seen later their presence would explain the double circulation postulated by Barcroft [6] However because they have been described practically every where else their existence in muscle is likely and suggestive Some authors notably Dible and Saunders have seen them [7]

Other Arterial Mechanisms

Certain communications between moderately large arteries are concerned with the equalization of blood pressure and the equable distribution of blood Among these are the loops between the branches of the mesenteric arteries, the loops between the palmar and digital arterial arcades and between the vessels making up the circle of Willis Reynolds[8] has described the spiral arteries in the ovaries of the rabbit and has analyzed the probable hemodynamic consequences of such an arrangement Clara[9] has studied the pattern of helicine arteries (Rankenarterien ') of the corpora cavernosa

The spiral arteries in the uterus of primates need special mention since they form the basis of current explanations of menstruation and implanta

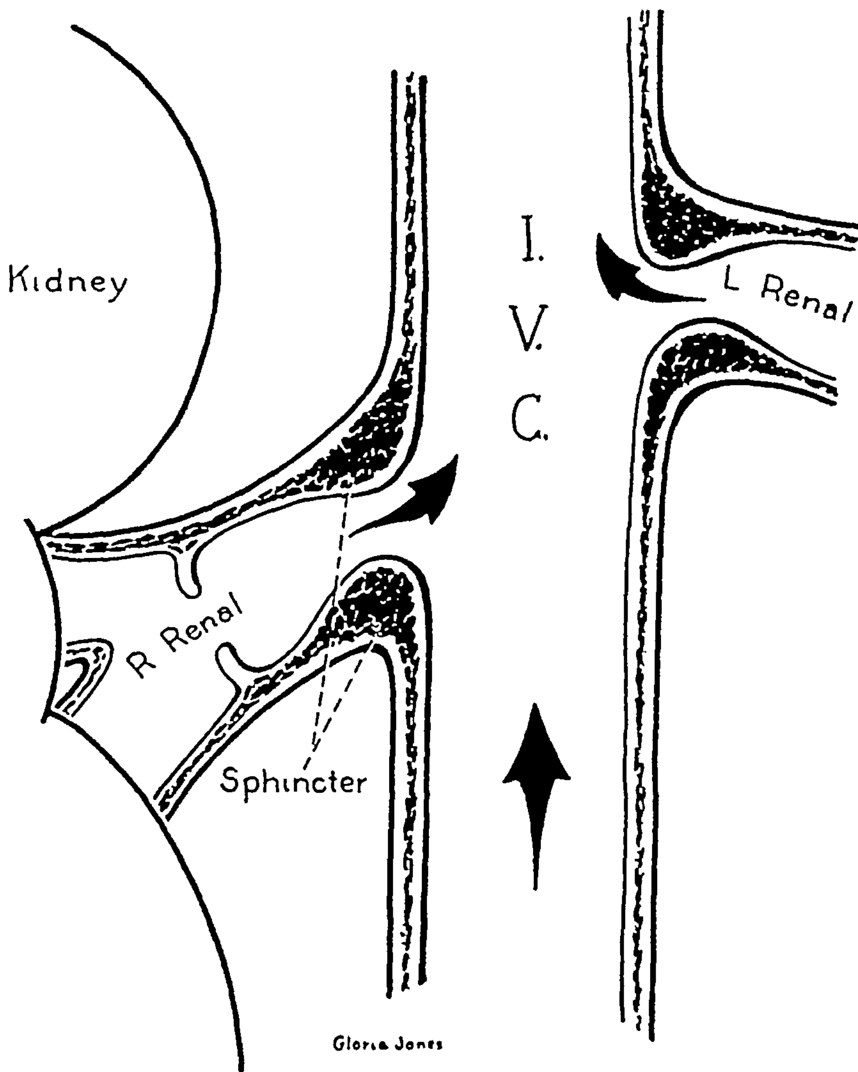


FIG 5 Muscular sphincters, consisting of a local increase of smooth muscle at the entrance of tributaries into a collecting vein Such sphincters are highly developed in lower animals and in the human fetus They decrease in number and atrophy with growth and aging in the human adult They have been recognized in the splenic, hepatic and renal veins Such muscular cushions have also been described at the origin of some visceral arteries They are likely to be under hormonal and neurogenic control. (Redrawn from Burch, G E A Primer of Venous Pressure Lea and Febiger, Philadelphia, 1950)

tion of the ovum All these mechanisms, which are usually ignored in the vascular tree of the extremities, give a vivid idea of the active nature of b'ood vessels in the living body They should stimulate detailed anatomic study of peripheral vessels, where they are largely unknown

Cushions and ridges are recognized histologically Their pharmacologic hormonal and neurogenic control needs much more investigation. Special arterial sphincters have been described in the spleen, in the liver, in the helicine arteries of the erectile apparatus and quite recently in the kidney Sphincters have also been described in veins. In the cavernous erectile tissue of the penis and clitoris the draining veins possess a well developed musculature, apparently in order to keep the blood within the cavernous tissue during erection The nervous control of this mechanism is still unsolved, but axon reflexes may well be involved, uninfluenced by sympathetic denervation

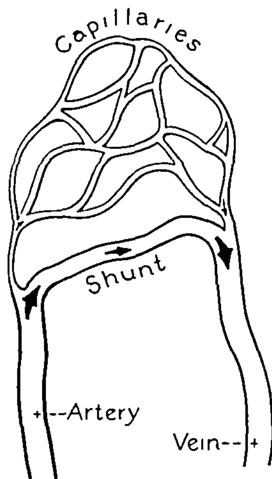


FIG 6 Preferential channels, "thoroughfare" vessels, in the precapillary circulation. These shunts permit more direct transmission of pressure from the arterial to the venous system They offer less resistance to flow than the capillaries, and help maintain venous pressure and flow (Burch G E. A Primer of Venous Pressure Lea and Febiger Philadelphia, 1950)

STRUCTURES WITH DUAL BLOOD SUPPLY The liver the lungs and in certain vertebrates, the kidneys have a blood supply from two sources The relationships between them in physiologic and pathologic states are of great interest Shifting of blood from one system to another accomplished by segmental occlusions or nervous regulation may materially enhance the function of the affected organ

Capillary Bed

The concept of capillary contractability has undergone revision A mode of closure by "swelling of the endothelial cells particularly of the nuclei which round up and bulge in the lumen has been suggested by many workers An excellent review on the structure of the blood capillaries has been made by Danielli and Stock [10] Hormonal changes in capillary permeability have recently been stressed by Kramar and his coworkers[11] who feel that a cortico-adrenal response to stress is manifested in permeability changes Nerve endings in capillary walls have not been seen but fine filaments of nerves can be stained with intravital methylene blue

CONTROL OF REGIONAL VASCULAR MECHANISMS

This brief description only serves to emphasize that vasoconstriction and vasodilation of the arterioles is not the sole mechanism of regional dis-

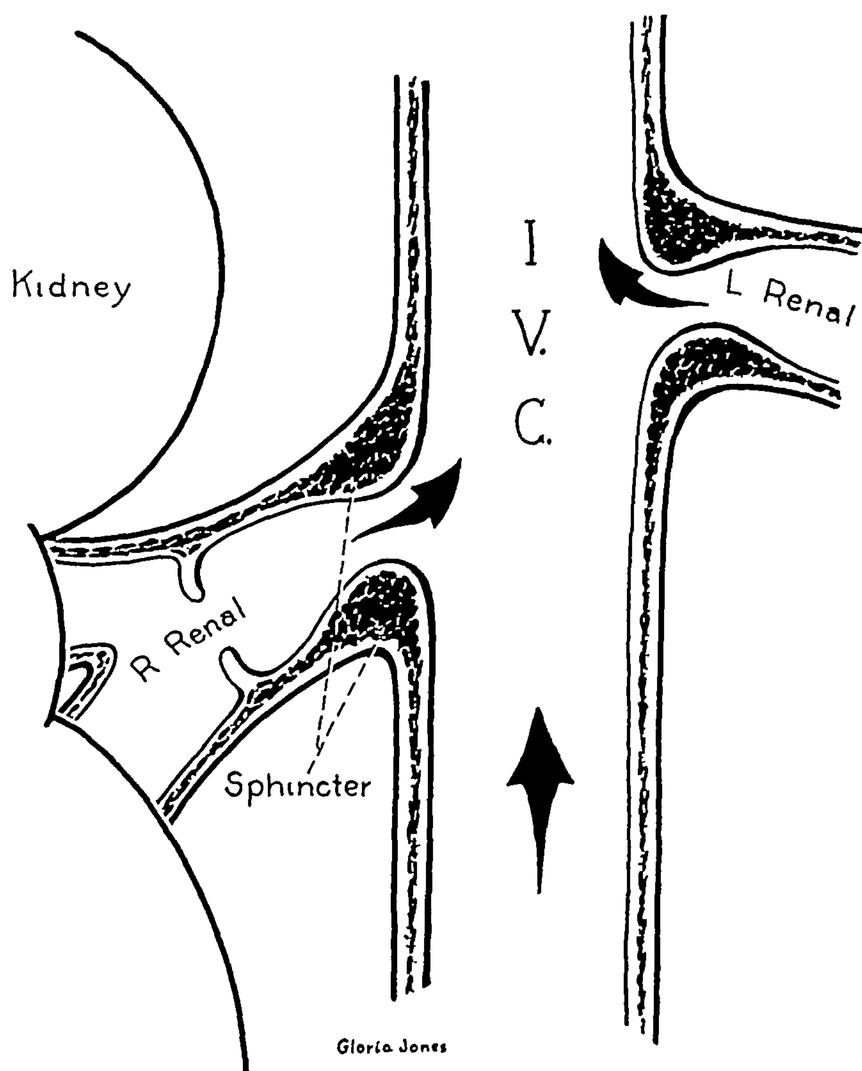


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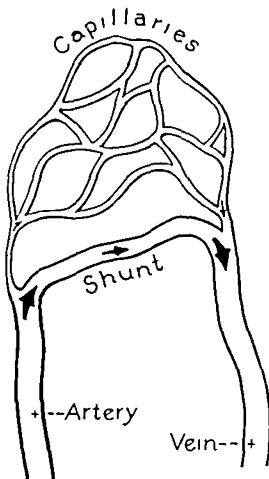


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CONTROL OF REGIONAL VASCULAR MECHANISMS

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tribution of blood Shunts, bypasses and alternate pathways operate to distribute the blood according to needs As with other effector mechanisms their control is dual nervous and hormonal Since surgical procedures may effectively or harmfully interfere with these delicate adjustments, existing knowledge on this seldom discussed aspect of peripheral circulation may be briefly summarized

In the first place, the typical arteriovenous anastomoses, from which the glomus tumors originate, are richly endowed with heavily myelinated and fine nonmyelinated fibers The former constitute an afferent, sensory nerve supply, through which painful stimuli and reflex activity may originate

In the case of I L A (Ill Res and Ed Hosp , #6937), for a cold, painful, swollen, hyperhidrotic hand, which developed following injury, a dorsal sympathectomy was performed with the erroneous diagnosis of a causalgic state The edema, sweating and vasoconstriction disappeared, but a small well localized painful subungual glomus tumor now became evident Removal of the pinpoint-sized, bluish mass eliminated all complaints This human experiment demonstrated the dual nerve supply of this type of arteriovenous shunt since first the efferent and then the afferent pathway was interrupted

The second pertinent observation refers to the oxygenation of venous blood which occurs shortly after sympathectomy or sympathetic block (Table I)

Table I

THE EFFECT OF SYMPATHECTOMY ON VENOUS OXYGEN SATURATION*

PATIENT	O_2 SATURATION PER CENT
(1) Operated side	82 0
Control side	30 6
(2) Preoperative	62 9
Postoperative, 12th day	77 4
Postoperative, 56th day	79 0
Unoperated side	44 0
(3) Preoperative	77 4
Postoperative	85 7
(4) Operated side	76 2
Unoperated side	58 4
(5) Operated side	73 6
Unoperated side	42 5

Average rise in O_2 saturation, 34 per cent

* Hick, F A , and de Takats, G , 1936

All determinations were run in duplicate Cases number 1 and 3 had cervicodorsal sympathectomies Case number 2 had lumbar sympathectomy All three patients had thromboangiitis obliterans The venous oxygen saturation and its percentual rise following sympathectomy closely parallels the clinical improvement

The magnitude of the rise of O_2 content suggests the opening of A V shunts following sympathetic paralysis Whether this is a temporary or a

permanent effect eludes me Freeman[1] has felt that such diversion of blood from the capillary bed through arteriovenous shunts is responsible for the paradoxical gangrene after sympathectomy This phenomenon will be dis cussed later in part III

A further effect of sympathetic denervation on the shunt mechanisms is that it changes the pressure gradient so that blood now encounters less re sistance in the alternate pathways and even those that have not been func tioning can now come into play The diagram of Wiggers (fig 7) graphically

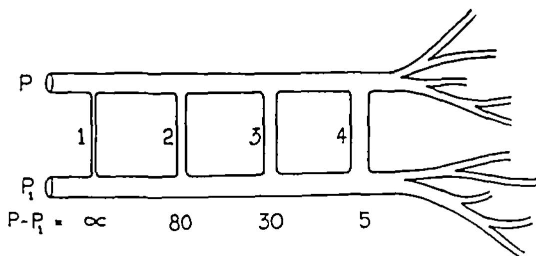


FIG 7 Diagram illustrating hydraulic principles which determine flow through side channels in tubes of different size Two vessels, P and P_1 are shown to communicate through four channels. Differential pressures of 5 30 and 80 mm. of mercury are required to move blood through the collateral channels 4 3 and 2, respectively An infinite pressure would be required to send blood through channel 1 The importance of head pressure and the diameter of collaterals is obvious. (Wiggers, C J Problem of Functional Coronary Collaterals. Exper Med. and Surg. 8 402, 1950)

illustrates the importance of pressure and diameter in alternate pathways As will be pointed out in the next chapter sympathectomy measurably de creases collateral resistance[13] and this must contribute to the opening of shunts The shunts are exceedingly sensitive to sympathetic stimuli and a minimal stimulus closes them before it affects the arteriole (R T Grant[1]) On the other hand it is possible as suggested by Walder [1] that increase in capillary resistance forces the blood (or microscopic glass-spheres in the animal experiment) through the shunts without any active mechanism in the shunt itself There is a curious epitheloid structure in the middle of the shunt which must have a function [5] On the other hand Burton believes that flow through the A V shunts is regulated by the pressure and vasomotor tone prevailing at the point where they join the artery From observations of the rabbit s ear in a transparent chamber it would seem that many stimuli affect the shunts and that vasomotor paralysis opens them until such time when the capillaries regain their tone

Our knowledge is even less definite about the hormonal regulation of the bypasses The coiled arteries in the ovary as studied by Reynolds extend straight out and then completely open up under gonadotrophin [1] This is one

of the functions of the spiral vessel, namely, to permit adaptation of the artery to a change in size of the ovary But the spiral vessels also serve for a localized reduction in blood pressure, just as coils are used by heating engineers to drop the pressure [1] Epinephrine has been studied extensively in its relation to the terminal vascular bed, since the discovery of the physiologic action of norepinephrine, many of its actions have to be restudied and reinterpreted Little is known about the combined action of these substances on the shunts, but the idea that capillary contraction would open the shunts by purely physical means is intriguing Should this be verified, then medullary adrenal secretion would maintain patent shunts by closing capillaries This would also apply to the posterior pituitary secretion, whose physiologic action in the adaptation syndrome, after exercise and in producing pallor in fainting, have received increased attention [14]

COMMENT

The question naturally arises whether or not vascular surgery can in any way benefit from this recent interest in shunts Their nervous and hormonal control is clearly in an early phase of study Yet, as will be shown in the next chapter, some effects of sympathetic paralysis may have definite influence on these shunts, especially in interpreting the response of muscle to this operation [6]

One is also in doubt as to how much the functional and organic vascular diseases affect the shunt While Popoff described degenerative changes in the A V anastomoses,[15] the recent observations of Dible are very encouraging for the future of vascular surgery Dible believes,[7] mostly on the basis of radiopaque injections of the amputated limbs, that the vessels of the foot, even in cases of total obstruction of the main arteries, can be well visualized, that their condition is surprisingly excellent and that the ischemia leading to arteriosclerotic gangrene is due to stagnation, lack of arterial inflow, and not to terminal occlusion This finding is important in justifying attempts of revascularizing arteriosclerotic extremities [16]

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CHAPTER 3

VASOMOTOR APPARATUS

A FORMAL DESCRIPTION OF THE ANATOMIC PATTERN, PHYSIOLOGY AND PHARmacology of the vasomotor system has been adequately given in so many monographs that it will not be repeated here, except to refer to the writings of Bayliss,[1] White, Smithwick and Simeone,[2] and Krieg [3] The surgical approach to the ganglionated trunks will be given in part IV, Surgical Technique In this chapter, the present thought on vasomotor function under normal and pathologic conditions will be described, with obvious emphasis on the effect of sympathectomies on human circulation

The concept of regional sympathetic denervation for organic or functional vascular disease was simple enough when it was first undertaken by Adson and Brown in this country[4] and Diez[5] in South America Prior to that time, Leriche had advocated stripping the adventitia of arteries,[6] and later extended this operation to include the ganglionated trunks There is still much emphasis in many quarters on the "release of vasospasm" by sympathectomy, although we have repeatedly pointed out that while intermittent or continuous vessel spasm does exist, the majority of sympathectomies will interrupt normal vasomotor tone and produce vasomotor palsy which is beneficial in many regional circulatory disturbances [7] In fact, it is in such cases that sympathectomy is most beneficial

Vasomotor tone varies among individuals and in the same individual, and is under the influence of many factors High vasomotor reactivity is characteristic of many persons in a prehypertensive state The work of Hines has unquestionably demonstrated a higher incidence of vasomotor reactivity in the children of hypertensive parents [8] This is best measured by the cold-pressor test, but standard emotional stimuli or the breathing of 5 to 7 per cent carbon dioxide can also serve in studying the state of the vasomotor center.[9] Cold, fright, pain and prolonged emotional tension bring on the first stage of the adaptation syndrome of Selye, *i e*, the alarm reaction [10] The vasomotor center here is bombarded by central and reflex stimuli, and by the mobilization of epinephrine, norepinephrine and the posterior pituitary substance These hormones have a central and peripheral site of action, often the central and peripheral actions are antagonistic to each other and can only be demonstrated when one of them is eliminated

Thus, Gellhorn and Steck[11] showed on some of our sympathectomized patients that carbon dioxide, normally a centrally acting vasoconstrictor agent, dilates the terminal vascular bed in sympathectomized limbs The

principle of dual action of the autonomic nervous system has not been sufficiently explored in explaining some of the sequelae of sympathectomy Thus, unopposed vasodilation brought on by stimulation of parasympathetic vasodilators must be distinguished from abolition of sympathetic vasoconstriction one of the results of sympathectomy

For a considerable time the existence of sympathetic vasodilator fibers in man had been seriously questioned As pointed out by J H Burn[12] many years ago vasomotor studies were dominated by cat physiology but should the hare for instance have been chosen as an experimental animal our concept of sympathetic stimulation might have been vastly different Today with the advent of techniques applicable to human physiology, observations on man are the best possible approach to this problem Furthermore with the recognition that cholinergic and adrenergic fibers secrete dilator and constrictor substances and that the tissues or vessels themselves contain antagonizing enzymes the basis for nervous stimulation is more and more transferred to the nerve endings and to the chemistry of their environment[13]

INTERRUPTION OF VASOMOTOR FIBERS IN MAN

The existence of three types of vasomotor fibers in man seems certain These are (1) vasoconstrictor fibers with a synapse in the lateral horn and in the sympathetic ganglion which produce efferent vasoconstriction (2) vasodilator fibers running in the sympathetic outflow from the cord and demonstrable in their effect on muscle circulation (3) vasodilator fibers in the posterior root system the antidromic fibers of Bayliss which conduct vasodilator impulses in retrograde manner through sensory afferents Their importance in causalgic state has been postulated[14,15]

1 Sympathetic Vasoconstrictors

Following sympathetic denervation one observes *early* and *late* changes It has been known ever since Goltz and Freusberg (1874)[16] that a maximal vasodilation occurs immediately after denervation but lasts only a few days In fact our experience no doubt as that of others, indicates that a warming of the extremity with pink digits and bounding pulses may be delayed to the *second postoperative day* then to subside again *five to six days later* The most accurate record of changes in blood flow obtained on limbs sympathectomized for hyperhidrosis or deep venous thrombosis in the presence of a normal arterial tree were presented by Walker Lynn and Barcroft[17] (fig 8) The blood flow increased an average of eightfold in the hands and about fourfold in the feet the maximum was reached on the second day and then quickly fell in the next two weeks The final flow two to three months later was about double the preoperative one A portable air plethysmograph was used for these studies At the same time the finger and toe temperatures rose and did not seem to fall concomitantly with the decrease in blood flow We

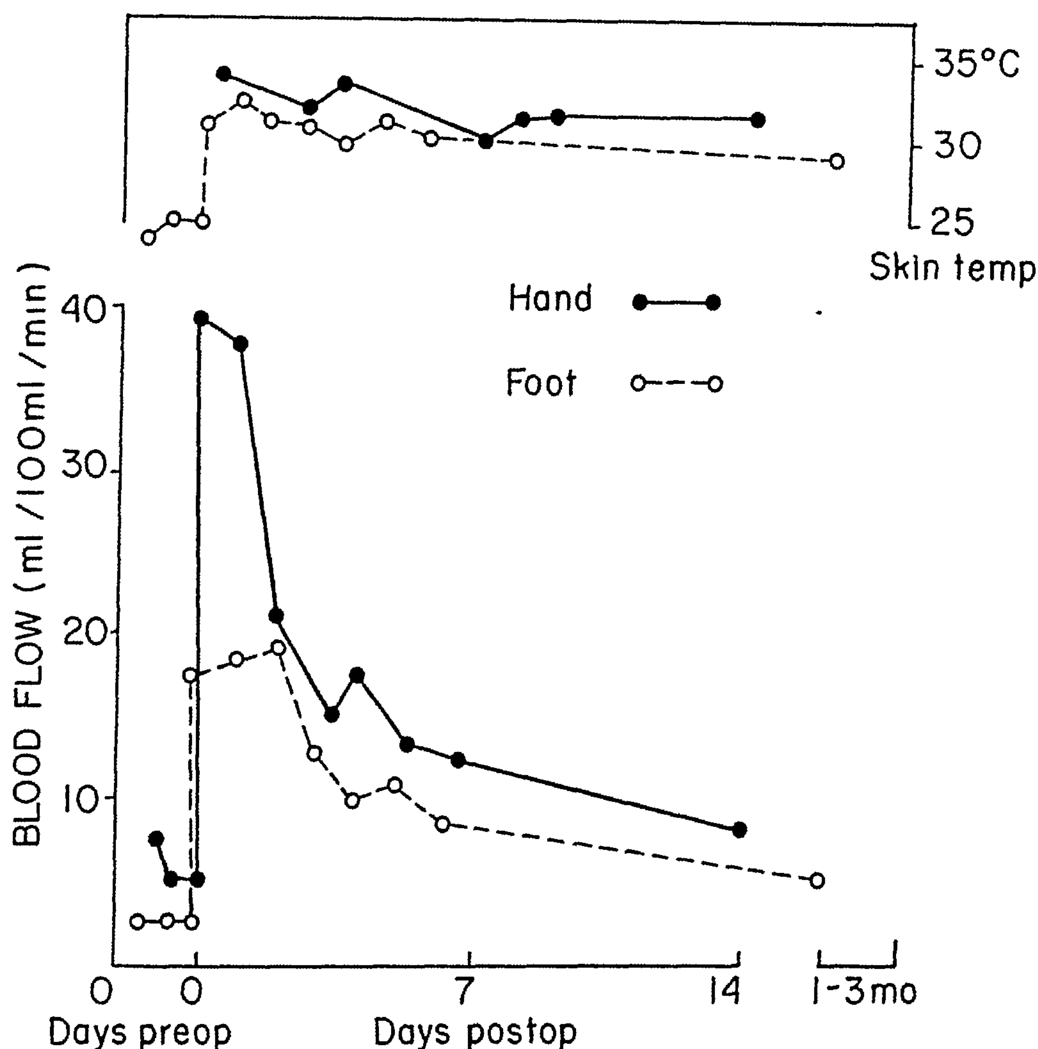


FIG. 8 Changes in blood flow following sympathectomy compared with the rise in skin temperatures. Straight lines refer to the hands, interrupted lines to the feet. Skin temperatures are on top, blood flows below Note the abrupt rise in blood flow within a day or two following operation, which decreases to about double of the preoperative level Skin temperatures remain elevated (Redrawn from Walker, Lynn and Barcroft St Thomas's Hosp Rep, *6* 18, 1950)

have seen a large number of patients subjected to lumbar sympathectomy for thromboangiitis and arteriosclerosis obliterans, in whom the toe temperatures on the operated side stayed 6 to 8 degrees F higher than the ones on the unoperated side, the longest observation was 25 years.

In the experience of the group at St Thomas's Hospital[17] there seemed to be no appreciable difference between a pre- and postganglionic section as far as the rate of reduction of blood flow in the first two weeks was concerned For Raynaud's phenomena, our group has undertaken the same type of experiments, doing an upper dorsal ganglionectomy for one side and a preganglionic section, leaving the ganglia intact, on the other No appreciable change, except possibly an earlier appearance of sympathetic vasoconstriction in the arm and hand, were observed on the side in which the ganglia were left intact

The vascular response to sympathectomy is far greater in the hand than in the forearm, a fact that had been emphasized by the observations of

Abramson and Ferris[18] (1940) and re-examined by Duff[19] The vascular re sponses in the hand and foot are essentially those of the cutaneous b'ood vessels whereas the forearm and the calf contain a great deal of muscle The increase in blood flow to the forearm was reached earlier than in the hand its extent was not only smaller but it returned to the preoperative level at a time (fourth to eighth day) when the blood flow to the hand was still quite high Similar results were obtained in the calf by Dornhorst,[20] who found that the maximum calf blood flow occurred on the day of operation This early return of blood flow to slightly above the preoperative is commonly explained by the *regain of intrinsic tone* In studying this mechanism Cannon and Rosenblueth [1] have stated that the effect of denervation extends beyond the distal neurons to the effector cells such as the striated, the smooth and the cardiac muscle cell They become increasingly sensitive to chemical stimu lation Supersensitivity is less marked if the section occurs through the penultimate neuron *i e* if the section is preganglionic As pointed out by Barcroft and Swan [20] the rate of regain of tone is so similar to the rate of development of epinephrine sensitivity that the events they represent must be closely related Barcroft and Swan listed the following hypotheses which have been suggested to explain the return of tone

(1) The arteries may become hypersensitive to circulating epinephrine [22]
(2) Hypersensitivity may occur to an unknown hormone [23]
(3) Intrinsic changes in the smooth muscle are responsible [24]

There is no certain proof for any of these assumptions The recent studies on the enzyme content of the arterial wall and its change after sympa thectomy may be another approach to the problem [25] It is important how ever for anyone performing sympathectomies to be aware of the regain of tone and of the decrease in the initially greatly increased blood flow Need less to say this return of blood flow to the double of the preoperative level might just make the difference between survival of the limb or gangrene It is also to be remembered that such blood flows have been obtained in a rest ing or basal state and that vasoconstriction from *erect posture reflex cold* and an *emotional state* is eliminated from the sympathectomized limb A sympathectomized extremity is thus kept as if its possessor were *horizontal in bed, wrapped in cotton* or in the *southernmost tip of Florida* where the aver age temperature is 85° F and *freed from all annoyance* it is kept as if its possessor were in a continuous state of euphoria or under the influence of hypothalamic depressants such as phenobarbital or Thorazine

2 Sympathetic Vasodilators

There has been much argument in the past about the mechanism of vasodilation occurring in skeletal muscle after the release of epinephrine or during a faint is it due to a release of vasomotor tone (inhibition of vaso-constrictors) or due to an active sympathetic vasodilation? Barcroft and Edholm[26] showed conclusively that the increase in the blood flow was due to excitation of sympathetic vasodilator fibers. Vasodilation was absent in

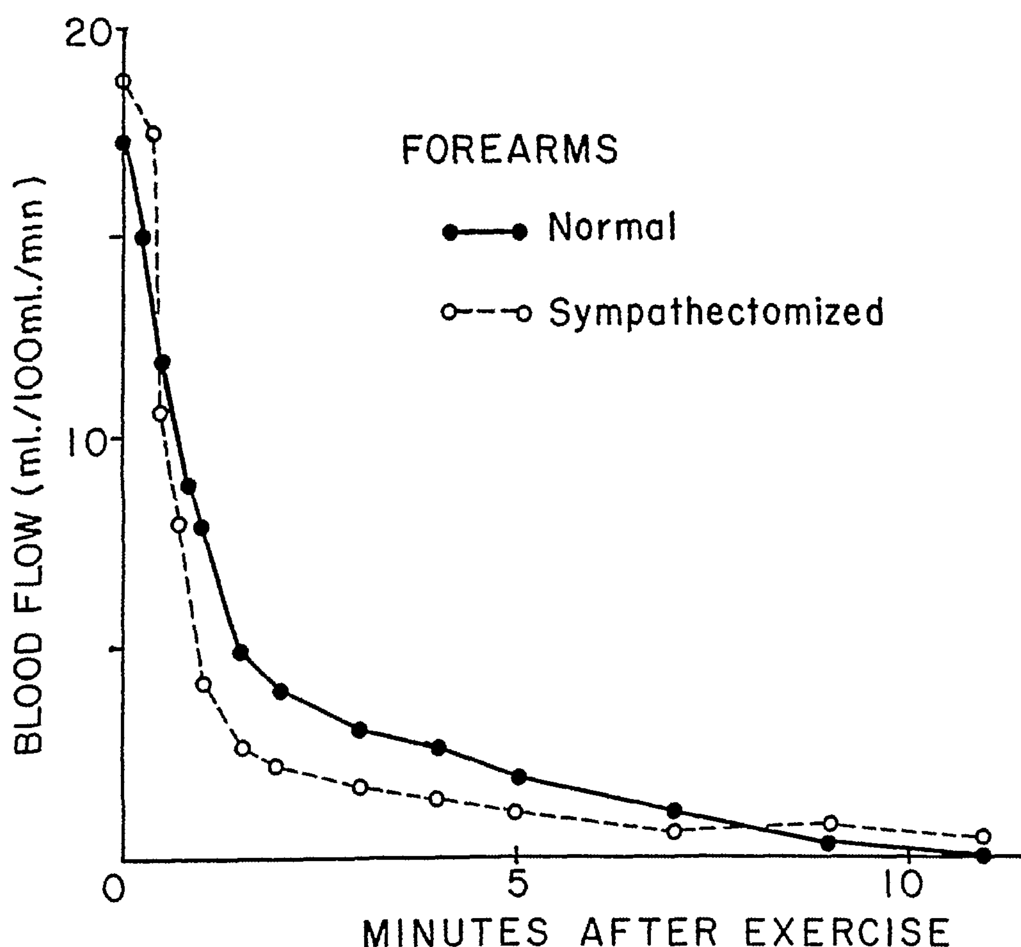


FIG 9 Blood flow following exercise in the normal and sympathectomized forearms. The hyperemia in the normal and sympathectomized muscle is similar (Grant, R T. and Pearson, R S B Blood Circulation in Human Limb Clin Sc, *3* 119, 1938)

the sympathectomized limb or after acute denervation of the muscles by nerve block Detailed evidence that it was not due to release of epinephrine or to sympathetic inhibition is given in the monograph of Barcroft and Swan [20] What is of great interest to us is whether or not sympathectomy inhibits active vasodilation in the muscle on exercise, since, if this were the case, the circulatory status of the limb would be harmed instead of improved after sympathectomy Such mutterings have been heard from the stronghold of certain internists

Grant (1938)[23] had found that during exercise the vasodilation in normal and sympathectomized limbs was similar Since this is an important point, a graph (fig 9) showing Grant's result is reproduced. Recently, Barcroft and his associates[27] showed that the activity of the vasomotor center had no effect on the circulatory changes taking place during or after muscular activity, yet release of sympathetic vasoconstrictor tone definitely increased the blood flow through muscle This discrepancy led Barcroft to the hypothesis that two circulations exist in muscle (1) anastomotic, consisting of arteriovenous shunts and regulated by the vasomotor center, and (2) nutritive, consisting mostly of the capillary bed, which is controlled by metabolites and functions without nervous control (fig. 6). As pointed out by Bar-

croft the existence of these two circulations may explain a paradox in some of the recent studies on circulation in the muscle

The radioactive sodium method of Kety[78] showed no increase in the circulation in muscle after reflex heating or sympathetic block however plethysmographic studies show that this does occur It is possible [0] that the difference between these two methods is explained by the fact that the sod um clearance does not pick up an increased flow through the anastomotic chan nels From the standpoint of clinical experience there is no doubt that in mild early claudication sympathectomy results in definite improvement in walking ability perhaps because the muscle arteries are not involved Gener ally speaking, however sympathectomy will not improve claudication par ticularly if it is severe only restoration of major arterial pathways can do that It is also common observation that improvement after sympathectomy continues for about a year after the operation and blood flow stud es done immediately following lumbar block or sympathectomy may not reveal slowly progressive improvement

Thus one can say that the elimination of sympathetic vasodilators to muscle seems to be greatly offset by the release of vasoconstrictors Actually sympathetic vasodilators do not enter into the mechanisms which produce hyperemia in exercising muscle and vasoconstrictor tone predominates at least in temperate climates

3 Antidromic Vasodilation

It was Langley[1] who coined the word antidromic and in our studies on the causalgic state [9] we have used the concept that impulses passing along the sensory nerves in an *efferent* direction produce vasodilation The fibers responsible for this antidromic vasodilation were thought by Bayliss[30] to be anatomically indistinguishable from the ordinary sensory afferent fibers failing to degenerate when the roots are cut between the cord and the gan glion but degenerating when the dorsal root ganglia are removed The prob lem of antidromic conduction has been reinvestigated with more modern methods by Hinsey and Gasser [31] and Bishop et al [3] They recorded action potentials and found the fibers to be slow conducting and nonmyelinated, not concerned with any afferent function Bayliss[1] found that this mechanism was very sensitive to mechanical stimulation and that in a peripheral nerve where both sympathetic and sensory fibers are present one can encounter a stage of regeneration in which the posterior root fibers have regenerated but the sympathetic fibers have not In such a situation stimulation of the distal end of the radial nerve of the cat caused marked vasodilation Also, in a sympathectomized limb stimulation caused pure vascular dilatation There has been some suspicion about the occurrence of vasodilation following stimulation of the motor supply to an area but this possibility has been overwhelmingly discounted

That the axon reflex, which originates from an area of cutaneous stimu lation, spreads to the next division of the sensory nerve and produces flush

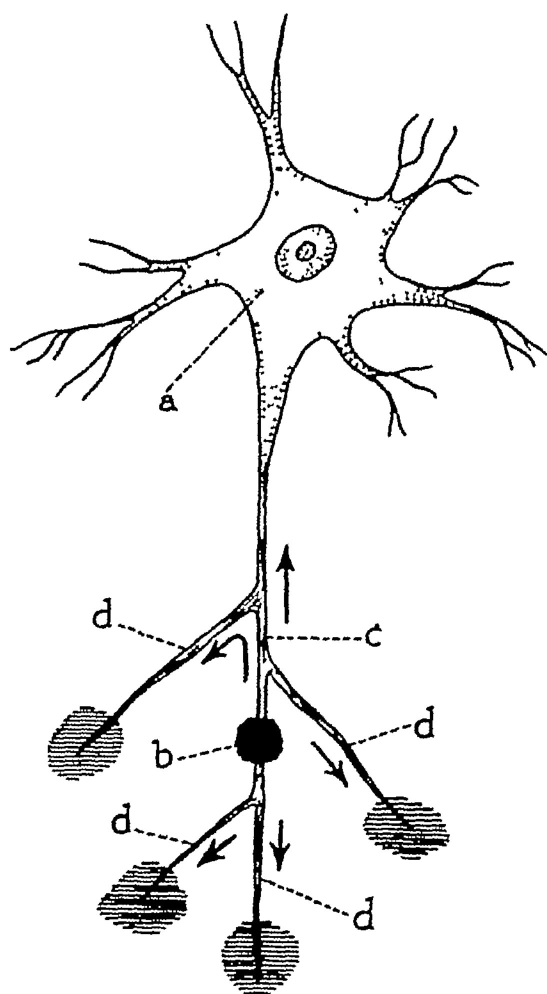


FIG 10 An axon reflex. The efferent arc utilizes a sensory somatic nerve in antidromic direction. (a) The ganglion cell, (b) area of stimulation, (c) the sensory afferent fiber, (d) the efferent vasodilator fiber Lightly shaded areas are diffusible substance at nerve endings.

ing of the skin by efferent impulse, is operating on the basis of the antidromic vasodilation is also a useful and satisfying hypothesis (fig 10). Visceral axon reflexes, operating in the lung, kidney and heart, may play an important role in vascular phenomena [33]

CHEMICAL TRANSMITTERS OF NERVOUS IMPULSES

The liberation of chemical substances at the nerve endings of cholinergic and adrenergic nerves (acetylcholine and norepinephrine) is inhibited by the enzymes cholinesterase and amine oxidase Both epinephrine and norepinephrine secreted at nerve endings on efferent sympathetic stimulation disappear rapidly on being exposed to amine oxidase From a clinical standpoint, the following observations of R H S Thompson[25] are of interest

(1) *Amine oxidase is found in greater concentration in the aorta, pulmonary and renal arteries than in the brachial, femoral and ear arteries.* This would mean that vasoconstrictor action is less opposed and thus more intense in the vessels of the limbs (2) *Sympathectomy does not produce a diminution of the epinephrine-oxidizing enzyme* and thus this enzyme behaves like cholinesterase does after denervation This might suggest that the amine oxidase is localized in the muscle cell, not on the nerve ending, and thus does not explain the increased epinephrine sensitivity following acute sympa-

thetic denervation (3) *Amine oxidase is unusually sensitive to slight reductions in oxygen tension* which may explain why ischemic fingers and toes show Raynaud s phenomena so easily Epinephrine or norepinephrine here can act far more unopposed than in a terminal vascular bed with a normal supply of oxygen No one has so far studied the possible increase of the amine oxidase in the ischemic digit after sympathectomy

THE LATE RESULTS OF SYMPATHECTOMY

The early changes after sympathectomy particularly the regain of tone together with the increased sensitivity to epinephrine have already been dis cussed In observing the late changes, Barcroft and Hamilton[34] assessed the completeness of sympathectomy with the plethysmograph using the principle of Landis and Gibbon in that the completely sympathectomized extremity warming of the body produces no increase in blood flow The blood flow at the end of the period was divided by the initial resting blood flow normally a 6 1 or 8 1 increase was found a heating ratio of 6 or 8 In a completely sympathectomized subject the heating ratio is approximately 1

All hands tested within six months of the denervation showed a heating ratio of 1 However when 16 hands of the 17 were tested one to one and one half years later, most of them showed evidence of some return of sympathetic activity This was also true of the return of sweating to some of the fingers a finger with a blocked ulnar nerve would react with no sudomotor activity Since some of our sympathectomies both lumbar and dorsal date back as long as 20 to 25 years we had ample opportunity to observe return of sweat ing under ordinary casual observation We can state with some assurance that in the lower extremity return of sweating is clinically not manifest un less of course, the lumbar sympathectomy was inadequate particularly sweating along the distribution of the femoral and saphenous nerves is always present when the first lumbar ganglion is not removed We have not found that the sweating area of the thigh or the band along the saphenous nerve would *extend* after a period of years although a *marginal hyperhidrosis* and pilomotor response has been frequently noted. On the upper extremity how ever as noted by all observers the reappearance of sympathetic activity is frequent and clinically noticeable in approximately 60 per cent of the cases Here again, since most surgeons do not wish to remove the stellate ganglion for reasons to be discussed later some of the fibers remain connected to the cord But even after evidence of complete denervation sweating, vasospasm and discoloration of fingers on exposure to cold return in over one half of the patients if they suffer from primary Raynaud s disease Interestingly enough, secondary Raynaud s phenomena accompanying thromboangitis frostbite or arteriosclerosis do not recur Activation of accessory sympathetic ganglia located in the anterior roots or in the cord first pointed out by Skoog,[35] may be responsible for this A central nervous stimulus or possibly a hormonal one may be operating in such cases of recurrence One may also recall that in essential hypertension, after splanchnic section and a type of dorsolumbar

sympathectomy which leaves the upper dorsal ganglia intact, coldness, tingling and even typical Raynaud's phenomena may develop in a few weeks in nondenervated areas This observation may indicate that the existing vasoconstrictor impulses and probably the sudomotor ones now spread over the nondenervated areas, causing hyperhidrosis and Raynaud's phenomena, *which were preoperatively absent* This is probably a thermoregulatory adjustment and has no bearing on a recurrence or progress of the vascular disease

The existence of actual regeneration of cut sympathetic pathways was forcibly brought home to us in 1939, when a 1 inch gap of a major splanchnic nerve was seen to have been bridged within a few weeks in the experimental animal [36] In man, however, excising long segments, placing metal clips on the cut ends and finding that the reconnections function poorly and maintain very slight vasomotor activity,[20] are sufficient guarantee that actual regeneration of cut fibers is of slight significance What does seem important is that if the diseased vascular state for which the sympathectomy is undertaken is of a central origin, the nervous impulses are more apt to find collateral pathways than if normal vasomotor tone is interrupted Should one assume that some vasospastic phenomena are due to hormonal activity, such as the adrenal denervations or partial adrenalectomies postulated by Leriche, then a sympathetic denervation may even aggravate the Raynaud's phenomena in the denervated area No one, to my knowledge, has observed such a case There is good evidence that the late changes after sympathectomy are due to hypersensitivity to pressor substances

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CHAPTER 4

BLOOD CLOTTING

EVERY SURGEON NECESSARILY MAINTAINS AN ACTIVE INTEREST IN THE PROBLEM of blood clotting How could he otherwise treat the thromboembolic phenomena following operation and the different forms of acute venous thrombosis which are so frequently encountered, especially in the aged?[1] The vascular surgeon, who opens and closes arteries, ligates and aspirates veins, extracts clots from accessible parts of the vascular tree and clamps major arteries during resection of diseased segments, has an added interest in the phenomenon of blood clotting In this chapter a brief summary of our present conception of thrombosis will be outlined, without getting lost in the maze of data which has accumulated in the literature on thrombosis. The annual conferences on *Blood Clotting and Allied Problems* sponsored by the Macy Foundation contain the latest thoughts on the subject [2]

Stasis has held the interest of pathologists and clinicians for a long time as the most important localizing factor in thrombosis Aschoff, in his *Lectures on Pathology*,[3] published a helpful diagram to illustrate the axial stream of the plasma and the sedimentation of the corpuscular elements when the flow of blood is retarded (fig 11) The recent intravital studies of Knisely and his co-workers[4] on the phenomenon of sludge may simply be a photographic registration of the slow stream and the sedimentation with loss of plasma It is certain that retarded flow, especially in the terminal vascular bed, also affects permeability, and thus the loss of plasma will result in hemoconcentration and clumping of cells. Stasis is purely a physical phenomenon, not affected by heparin, as Laufman and his co-workers have shown,[5] and is definitely reversible Nevertheless, it may be the forerunner of thrombosis since it adds to stagnation and since it agglutinates red cells and platelets

Aschoff[3] also discussed turbulence in his lectures and, truly enough, one sees thrombus formation not only in areas of stasis but at bifurcations and at the site of venous cusps A diagram shown in a paper some years ago illustrated our conception of the effect of constriction by ligaments or tendons on the venous return of the lower extremity[6] (fig. 12) The influence of sudden hypotension below a critical level, such as that which occurs during operation, in massive hemorrhage or in other shocklike states, on the incidence of venous thrombosis has been emphasized by Davis and his associates[7] and is certainly of clinical significance Acute arterial thromboses occurring after operation or childbirth may well be related to prolonged hypotension, these

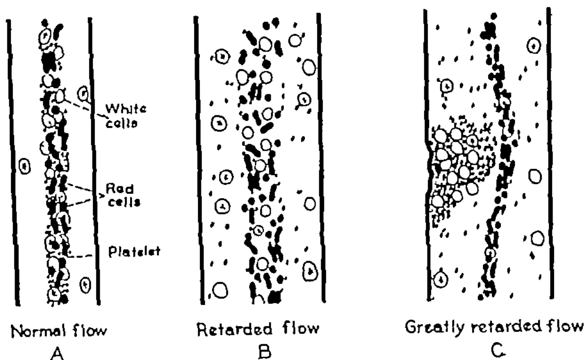


FIG 11 The pattern of blood elements in normal and retarded blood streams. During rapid normal flow (A) the marginal zone of the stream is mostly plasma, with only a few cells skimming the wall. In (B) depicting a retarded circulation, the axial stream consists mostly of red cells. Platelets and white blood cells have moved into the marginal zone. In (C) an almost complete standstill is shown. There is a bank of platelets in which white cells move slowly The axial current is slow and narrow

occur at the site of pre-existing stenoses and may be additionally triggered by changes in the clotting mechanism which follow such states

Chronic arterial stenosis or arterial aneurysms may be silent and undiagnosed for a long time A critical state of ischemia may be produced however by an occluding thrombus of the diseased vessel Outside of *trauma* whose effect on the vessel wall will be discussed shortly *hypotension* with its retarded flood flow is the most common cause of the superimposed thrombosis As pointed out by Edwards in his monograph [8] atheromatous arteries are frequently closed by thrombosis there is no evidence however that this was brought about by a tendency of the blood to clot Rather the volume of flow has been retarded to such a degree that an obstructive plug completes the occlusion Burton s diagram illustrates the relation between the velocity of flow and volume of flow which do not always parallel each other (fig 13) Stenosis and turbulence are particularly important in the arterial tree for the localization of a thrombus (fig. 11) whereas decreased velocity seems to operate in influencing the site of venous thrombosis

The clinical application of these considerations is obvious In a partially obstructed stenotic arterial tree or in a stagnant pool of venous plexus the critical factor maintaining patency is a well maintained blood pressure There is no evidence here of a disturbed clotting mechanism As discussed later the formation of the thrombus sets up a chain reaction and thus warrants the use of anticoagulants However a long prophylactive use of such drugs to

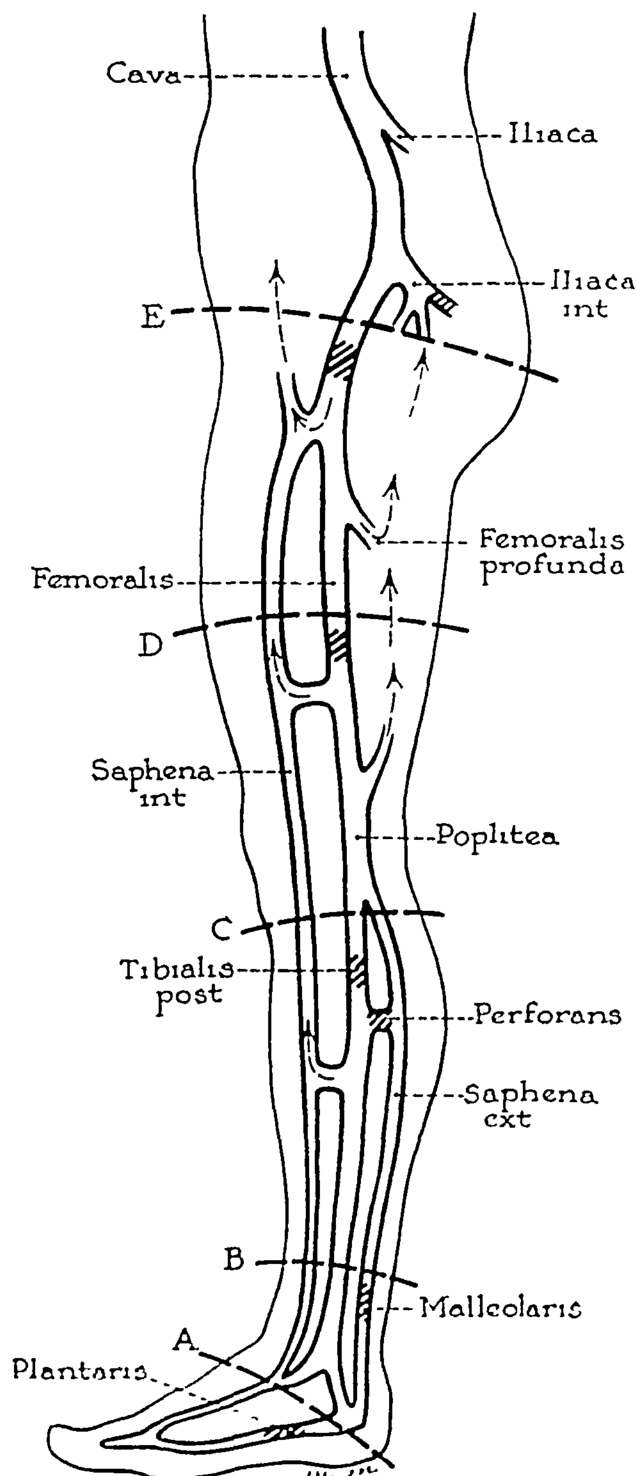


FIG. 12 Typical areas of initial thrombus formation in relation to constricting ligaments or tendons (A) tendon of the peroneus longus crossing the plantar vein, (B) upper border of the malleolar ligament, (C) upper edge of the soleus muscle, (D) adductor canal, (E) inguinal ligament (de Takats and Fowler Problem of Thrombo-embolism Surgery, *17* 153, 1945)

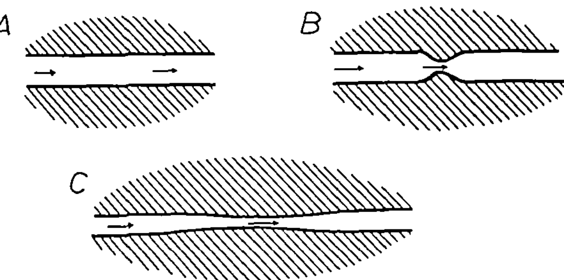


FIG 13 The relationship between velocity of blood flow and volume of blood flow A segmental stenosis (B) may increase velocity because of the reduction of the cross section of the area, but the volume flow decreases. On the other hand in a widespread, diffuse stenosis, or in a widespread vasoconstriction (C) the volume flow will decrease greatly but so will the velocity of flow (Burton, A C A Critical Survey of Methods Available For the Measurement of Human Peripheral Blood Flow Peripheral Circulation in Man A Ciba Foundation Symposium, Little Brown and Co Boston, 1954)

prevent cerebral or coronary vascular accidents is not our cup of tea and its value in spite of mass statistics [9] is not proven [10]

Temporary clamping of the aorta and its major branches especially in the presence of a diseased atheromatous peripheral vascular tree may lead to distal thrombosis and completely negates the benefits of a patent arterial transplant unless a distal run-off is controlled by regional heparinization This will be discussed as an important aid in restorative arterial surgery

THE STATE OF THE VESSEL WALL

Much has been made in the past of the fact that the normal endothelium of the vascular tree which is nonwettable [11] is one of the safeguards of the body against intravascular clotting. The sclerosing therapy of varicose veins rests on an inflammatory reaction of the intima to an irritant, causing the blood to clot when the intima is injured Ischemia of the vessel wall caused by ligature by a crushing clamp or by embolization to the vasa vasorum which occurrs in subacute bacterial endocarditis may cause thrombosis or aneurysm formation, depending on how much the elastica is damaged The role of the vasa vasorum their behaviour in atheromatosis has been the subject of much study [12] and the fact that the vessel wall becomes permeable to plasma under conditions of ischemia seems to permit the entrance of thromboplastic substances into the blood stream or plasma into the vessel wall, depending on filtration pressure While these substances can be readily washed away or neutralized, such a factor becomes important during conditions of localized stasis A periphlebitic vein may remain patent but will be

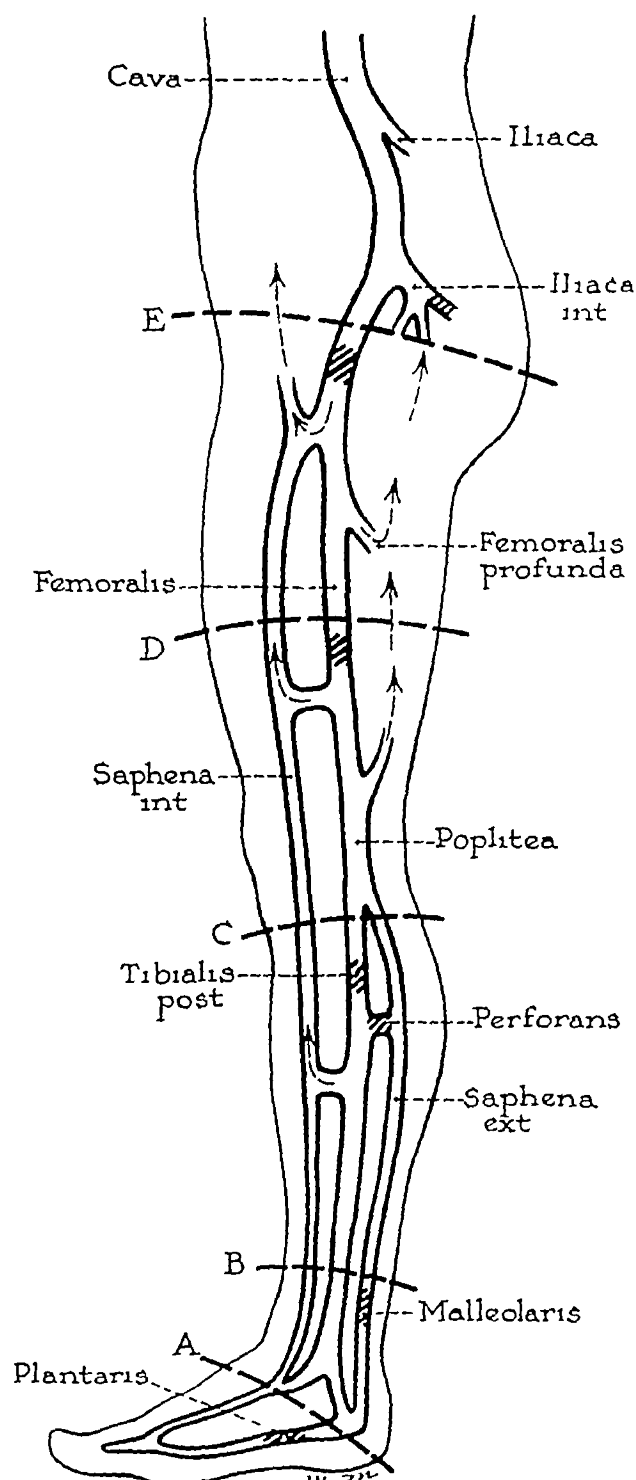


FIG 12 Typical areas of initial thrombus formation in relation to constricting ligaments or tendons (A) tendon of the peroneus longus crossing the plantar vein, (B) upper border of the malleolar ligament, (C) upper edge of the soleus muscle, (D) adductor canal, (E) inguinal ligament (de Takats and Fowler Problem of Thrombo-embolism Surgery, *17* 153, 1945)

The lining becomes well vascularized can be injected with India ink through the adventitia and truly becomes a functional intima even though it may not be endothelium (fig. 14)

The important point seems to be that the lining be vascular alive and not give off a negative electric charge which necrotic tissue does Such electrophysical considerations have been stressed by Sawyer and Deutch[13] and are capable of measurement Heavily radiated or sterilized tissues show no such electric potential and thus may be more suitable for transplantation than a homologous graft [14]

The interreaction between the blood and the wall of the vessel has been stressed by early students of thrombosis [15] The histologic studies of O Neill[16] and Samuels and Webster[17] indicate the adherence of platelets to the intercellular cement and the ease with which minor damage such as stasis or compression will start mural thrombi However as will be pointed out shortly, the blood is capable of dissolving small thrombi and that together with a certain velocity of flow will prevent a small mural thrombus (often a microthrombus only to be seen through the microscope) to become obstructive and thus clinically manifest

THE CLOTTING MECHANISM

Maintaining a constant fluidity of the blood is one of the homeostatic phenomena of the body We have used a diagram in the past to illustrate the balance of coagulant and anticoagulant factors[18] (fig 15) and have brought forth some evidence that such a balance may be readily upset by various

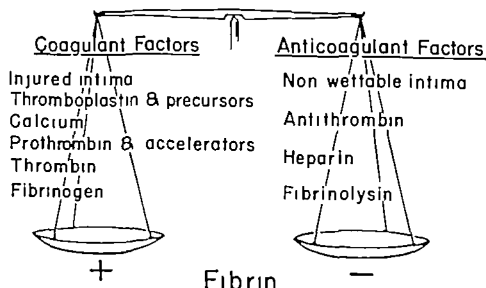


FIG 15 The clotting equilibrium, listing some of the known coagulant and anticoagulant factors in the balance. Clinical thrombosis occurs when the anticoagulant factors have been injured, depressed or exhausted. This is a homeostatic mechanism which is subjected to many stresses. (de Takats and Marshall Response of Clotting Mechanism to Postoperative Stress. Surgery *31* 13 1952.)

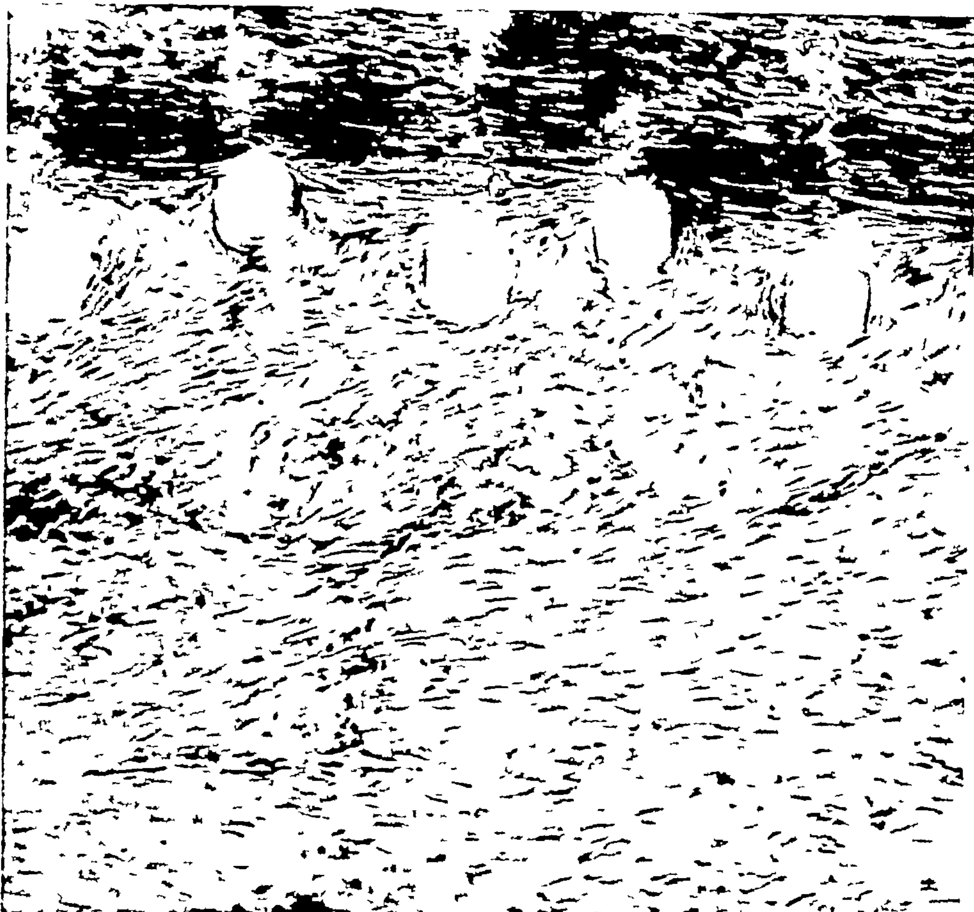


FIG 14 The "pseudo-intima" formed inside a transplanted tube of 200 gauge steel mesh On top is the new intima The circular defects are left by the filaments of steel Below is the fibrous but vascularized new adventitia (Lary, Meine and de Takats Experimental Use of Steel Mesh Tubes for Replacement of Arterial Segments A M A Arch Surg, *72* 69, 1956)

plugged by a thrombus when it occurs in a varix where pooling of blood takes place

Nothing militates more against thrombosis due to inflammatory or degenerative reaction of the wall than velocity of flow In an arteriosclerotic aorta, one can scrape away the intima, the subintimal atheroma and even part of the involved media only to find a line of cleavage A smooth, striated lining is left behind and, while a new pseudo-intima forms within a few weeks, arterial blood freely circulates in such a vessel and does not clot Whether the property of the ground substance, which stains with toluidine blue and is structurally close to heparin, has any influence here needs to be studied Nevertheless, the amazing fact remains that a vessel completely deprived of its intima can remain patent without any evidence of occlusion or mural thrombosis

The same consideration applies to the situation when arterial homografts, venous autografts or inert substances such as plastics, steel mesh or fiberglas are used to replace a resected arterial segment Although thrombosis may occur, this is mostly due to technical factors or to stenosis of the artery above or below the transplanted segment The nature of the new lining which forms in transplants and implants has been the subject of much recent study.

The factors one can recognize as producers of acute stress on the clot ting mechanism are fear anxiety pain cold hemorrhage and tissue trauma Such stress is ordinarily readily compensated by anticoagulant factors available to the body Heparin we know is liberated in anaphylactic shock but whether the human body really empties its heparinocytes under stress is now under investigation [19] There seems to be some evidence that the sudden disruption of mast cells liberates histamine and is part of the inflam matory reaction to injury Perhaps heparin is not liberated or is a general product of tissue breakdown [20] Once the clot is formed heparin will not dissolve it but it will prevent a spread of the thrombus in a distal and proxi mal direction it may also indirectly aid the dissolution of the clot by facilitat ing fibrinolysis Fibrinolysis, which may well be nature s most potent weapon in dissolving a clot is also regulated by the intensity of stress and is clinically obvious after massive hemorrhage When quantitative methods are used to determine the presence of fibrinolysin epinephrine fright and major surgi cal operations can be demonstrated to increase its value Cliffton and his as-sociates[21] have been especially active in dissolving 24 to 48 hour old clots in dogs and rabbits We have used aged plasma activated by trypsin in patients, injecting it above an arterial thrombus Dissolution of the clot was rapid in one case it occurred in the exposed artery under our own eyes It is more than likely that the widely used trypsin therapy is simply a nonspecific activator of fibrinolysin Intramuscular streptokinase may act the same way and so does any parenteral protein therapy

Naturally all these efforts must be controlled by some sort of determina tion of clotting and fibrinolytic activity The ordinary clotting time when blood is shed into a glass tube is a gross artefact tubes lined with Lucite or silicone are better but still do not represent conditions occurring in the rapidly flowing blood surrounded by endothelial lining. So at best, only a rough estimate can be made and in chapter 6 The Examination the capil lary the one tube venous and the heparin retarded clotting times have been listed Prothrombin times which occupy so many hours of work for the clinical laboratory do not, in my opinion, reflect the status of the clotting mechanism as a whole They are indispensable for following the coumarin type of anticoagulants all of which act on the prothrombin complex In chapter 12 Thromboembolism, the use of heparin the coumarins other prothrombin depressants and the fibrinolytic agents will be discussed Here it should only be pointed out that manifest clinical thrombosis can only occur when the body s natural defenses have been exhausted In other words intra vascular thrombosis is the decompensation of the clotting mechanism Ther apy directed against thrombosis needs only to restore the clotting equilibrium to normal by supplying the natural anticoagulants and fibrinolysins Chronic prothrombin depression produces an abnormal state which may in itself produce thrombosis on withdrawal.

Not enough emphasis has been placed in the past on the fact that a non treated or insufficiently treated acute thrombosis may develop into a chronic recurrent or even malignant type in which the slightest trauma, infection or

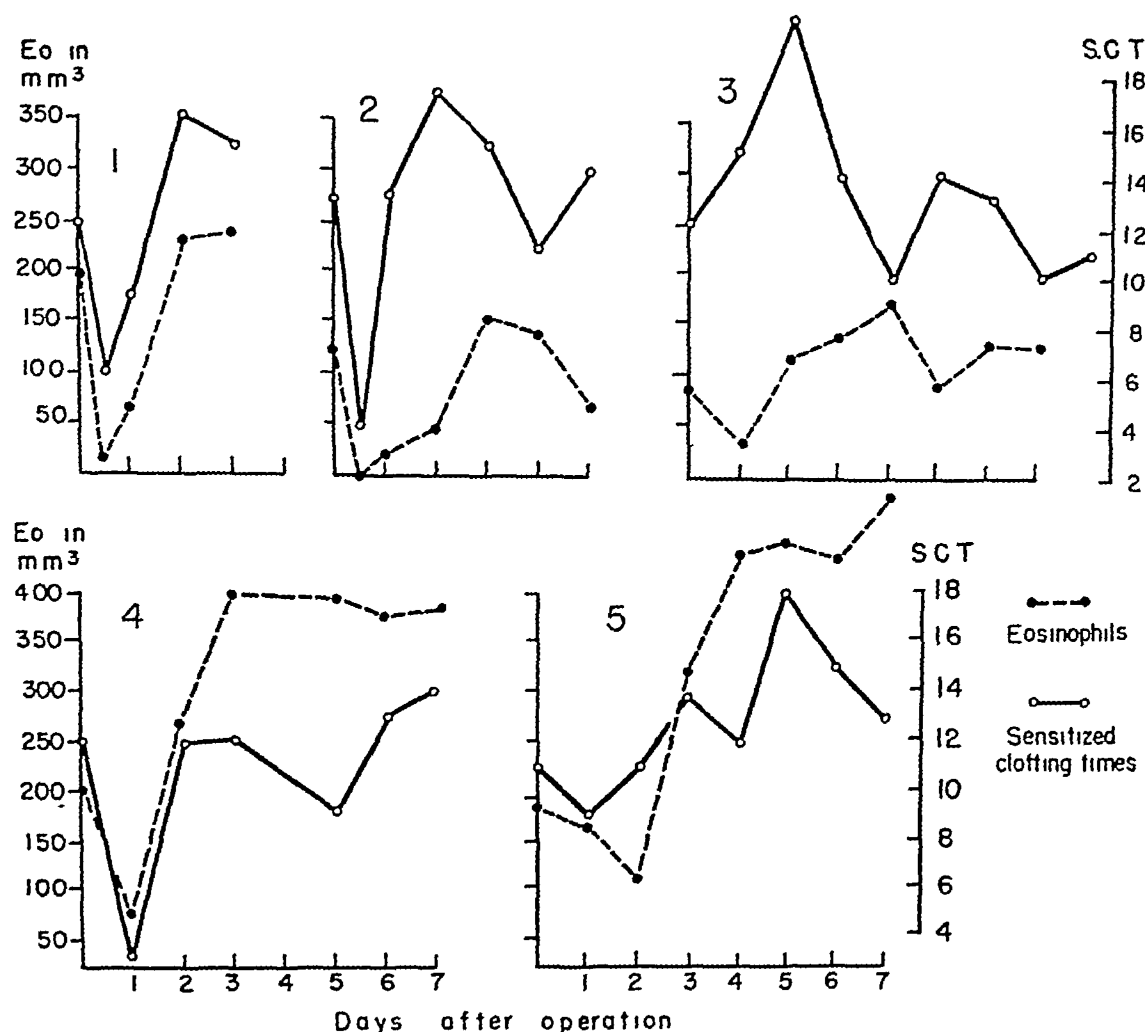


FIG 16 Simultaneous daily clotting times and eosinophil counts following operation (1) one stage Whipple operation for carcinoma of the pancreas, (2) right lumbodorsal sympathectomy for essential hypertension; (3) midmetatarsal amputation in Buerger's disease, (4) splanchnicectomy, second stage, for essential hypertension, (5) lumbar sympathectomy for diffuse arteriosclerosis In many hundred observations, a trend of parallel "stress curves" is unmistakable (de Takats and Marshall Response of Clotting Mechanism to Postoperative Stress Surgery, *31* 13, 1952)

stresses affecting the body as a whole Thus, an injury which liberates tissue juice sets up a chain reaction, consisting of several phases, which closely resembles, if it is not identical with, Selye's adaptation syndrome. The clotting times get shorter, then longer and then shorter again, parallel with the fluctuations in the eosinophil count (fig 16) One can plot such clotting curves following operations of different magnitude and find that the intensity of these fluctuations can be correlated with the magnitude of the operation Interestingly enough, the amount of heparin necessary to raise the coagulation time to approximately the double of the preinjection level also fluctuates during this period The more acute and widespread the thrombosis, the more heparin is needed to counteract the coagulant factors [18] The recognition of this principle led to the adoption of massive doses of heparin in the early phases of thrombosis, requiring a sudden and substantial reduction when the stage of convalescence sets in The practical application of this phenomenon will be discussed in chapter 12, Thromboembolism

propagated This is then a dynamic process not to be measured with a clotting time in a test tube

Voigt and I [72] therefore recently described a simple test which tries to convert these static tests to functional ones ACTH is given into the muscle in 25 mg. doses and heparin retarded clotting times are determined before and four hours after the injection The test has been devised to see what response if any can be elicited from the clotting mechanism after a minor stress

Normal young individuals respond with a prolongation of clotting time which is statistically significant (fig. 17) Many show no response, and some show a *shortening of the clotting time* The latter either have manifest thrombosis or are known to have a spreading carcinomatosis so that their normal defense seems exhausted Such patients are in need of prophylactic or therapeutic doses of anticoagulants In chapter 12, Thromboembolism, the practical application of these considerations will be elaborated

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CHANGE IN SENSITIZED CLOTTING TIME AND EOSINOPHILS IN YOUNG, HEALTHY, NORMAL SUBJECTS FOUR HOURS AFTER INTRAMUSCULAR INJECTION OF 25 mg

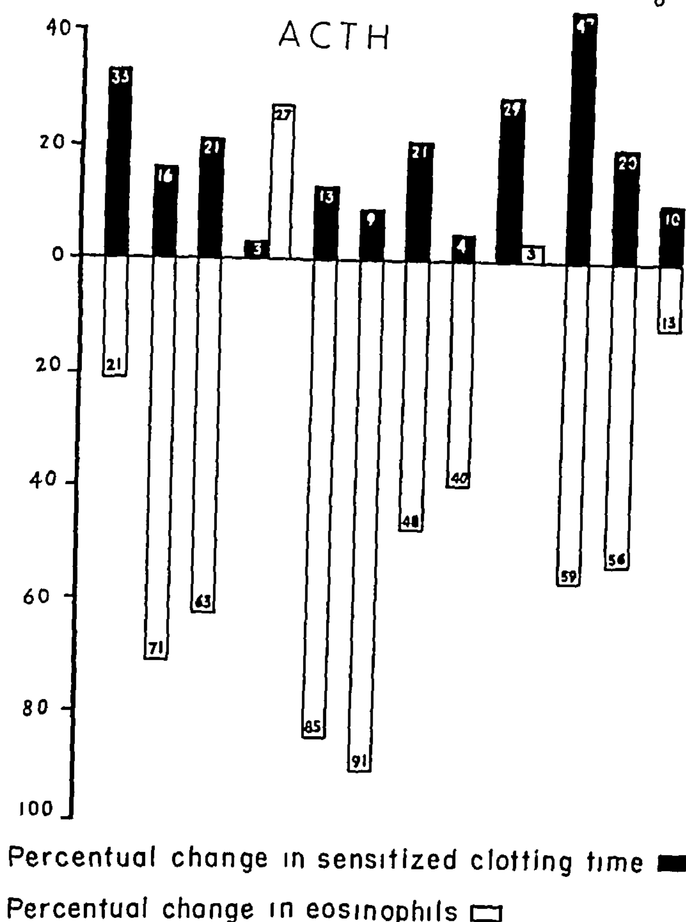


FIG 17 In twelve "normal," healthy subjects, 25 mgs of ACTH resulted in a prolongation of the clotting time A rise of 3 to 47 per cent of the preinjection level resulted The two minor rises occurred in menstruating women The drop in eosinophils also varied considerably The general trend of a longer clotting time with a drop in eosinophils is evident (de Takats and Voigt Response of Clotting Mechanism to ACTH Angiology, *4* 283, 1953)

surgical stress will initiate a blood clot Insufficient doses of heparin and long-term Dicoumarol therapy with taken doses may bring on such a smouldering state

The local thrombus, as Quick* described it so graphically, undergoes retraction and expresses a serum in which "rapid generation of thrombin occurs When the circulation is rapid, thrombin is quickly washed away and made innocuous by dilution If the blood stream is stagnant, the nascent thrombin clots the blood surrounding the thrombus This secondary clot becomes firmly attached to the first It contains entrapped platelets and will therefore retract By numerous repetitions of this process, the thrombus is

* Quick, A J Modern Concepts of Venous Thrombosis Practitioner, *166* 213, 1951

PART II

Methods of Diagnosis

The unity of all science consists alone in its method not in its material

KARL PEARSON *The Grammar of Science* 1937

21 Cliffton, E E , Grossi, C E and Cannamela, D Lysis of Thrombi Produced by Sodium Morrhuate in the Femoral Vein of Dogs by Human Plasmin (Fibrinolysin) Ann Surg , *139* 52, 1954
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PART II

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CHAPTER 5

THE INTERVIEW WITH THE PATIENT

A GOOD GENERAL HISTORY IS OBVIOUSLY A PREREQUISITE, BUT A CARDIOvascular history is of special importance since it alone frequently makes the diagnosis A few leading questions often direct the examiner into the proper channel so that additional data can be obtained and as a result special medical consultations can be sought In this chapter a few of these questions have been assembled together with an interpretation of their significance They contain much of what is usually discussed separately under past and present history symptoms and signs

When did you first notice the present symptoms? In congenital vascular anomalies a birthmark or an increase in the size of the limb may not be obvious at birth but is usually noticed during the first year If the lesion is due to trauma a pulsating hematoma or an aneurysm may not be detected for several weeks or months In fact, some arterial injuries may heal and remain quiescent for many years only to be activated by a fall a second trauma Symptoms of vascular insufficiency may start after a systemic infec tion such as subacute bacterial endocarditis pneumonia or rheumatic fever If the vascular lesion is more diffuse, ischemic symptoms may follow attacks of dizziness, chest pain nycturia and polyuria, or be concomitant with them If the lesion is on the venous side ask the patient if the venous dilatations developed after years of strain did they appear after childbirth after opera tions after a long bed rest with some swelling? Was there a charlie horse a cramping calf after these events? Or was there an infection of a toe an itching, scaly eczema a profuse bubbly vaginal discharge followed by a gradual increase of the size of the limb and a hard nonpitting swelling?

Was the onset sudden or gradual? Excluding the traumatic injuries a sudden vascular occlusion invariably means embolism or acute thrombosis The history of a sudden arterial occlusion always demands a study of the heart as a possible source of embolism this may be a congenital lesion a rheumatic heart with mitral stenosis and auricular fibrillation a subacute bacterial endocarditis, a myocardial infarction with mural thrombi in the ventricles very rarely a thrombus or a tumor in the pulmonary vein or a paradox embolus from the right side to the left. In patients with large ulcer ated atheromas of the abdominal aorta, a sessile thrombus may break loose

and obstruct some of the arteries in the lower extremities The sudden occlusion, however, may be due to an acute thrombosis at the site of a pre-existing vascular disease, thus a popliteal atheroma may have produced a gradual narrowing of the artery with no, or very vague, symptoms If the lesion ulcerates, if hemorrhage occurs into it[1] or if an active hypotension develops, a complete occlusion may take place giving rise to acute ischemia A non-traumatic popliteal aneurysm growing slowly may be asymptomatic until the sac suddenly clots, producing severe symptoms A patient suffering from thromboangiitis obliterans may have had some vague symptoms referable to his extremities or visceral organs, but a fresh occlusion of a major pathway or a closure of a functioning collateral vessel directs one's attention to the presence of vascular disease Haimovici[2] has shown that with up to a 70 per cent constriction of the lumen, arterial stenosis is asymptomatic

Venous occlusions are more often sudden, and may be traced to follow venous stasis, infections or changes in the clotting mechanism (chapter 12) Many patients have had previous episodes of deep venous thrombosis only to manifest themselves later by the development of a cutaneous pattern of veins, characteristic of collateral circulation

Lymphatic occlusions, when sudden, are usually infectious, although this obstructive lymphangitis may well be superimposed on venous occlusions, congenital lymphatic anomalies or malignant obstructions in the lymph nodes

Is there numbness or cramping on walking and where does it start? Is it in the bottom of the foot, which indicates a posterior tibial or plantar artery occlusion, is it in the anterior tibial compartment, which one sees after trauma or thrombotic occlusion of the anterior tibial artery, or, is it in the calves, which locates the occlusion to lower femoral or popliteal levels? Is it in the posterior part of the thigh, in the buttocks or the small of the back, which denote a high iliac or aortic obstruction? Of course, these symptoms cannot always be strictly separated from each other, but the *plantar* and *gluteal* ischemias are quite distinct and have localizing value

Is the vascular occlusion limited to one extremity? Is it more diffuse? Again, excluding the traumatic etiology, vascular disease often affects several parts of the vascular tree A patient with a pulseless foot, complaining only of that limb, may have occlusions in his other foot, at his wrist, or in his cerebral, coronary, renal or mesenteric vessels A good history may elicit suggestive symptoms confirmed by examination Occasionally the patient may have had an episode 20 years before the present onset of symptoms in the opposite extremity, which he had completely forgotten, such a history is suspicious of a "burned-out" but recurring thromboangiitis obliterans Then again, a patient with occluded arteries in both lower extremities may not volunteer symptoms of angina on effort since he cannot walk fast or far enough Careful questioning can elicit some symptoms following heavy meals, ingestion of cold drinks, exposure to cold air or emotional upsets He may be dizzy on rising, forgetful, or have had a change in personality due to a small subclinical vascular occlusion in the brain He may have a slight gnawing

ache in his abdomen aggravated by meals and perhaps a pain radiating to the back or in the left flank Abdominal angina due to sclerosis of the aorta and its branches or abdominal aneurysm are not infrequent

Such examples may be multiplied by many others they simply serve to illustrate the importance of thinking of the vascular tree as a whole when one of its segments is obviously affected

Are the symptoms worse at night during rest or are they aggravated by standing by walking by exercise? Cramping on walking (intermittent claudication) is of course the classic symptom of arterial insufficiency The reduced flow of blood to an extremity may suffice at rest but not when contracting muscles demand as much as a thirtyfold increase of blood *Pain at rest* if claudication is present is a sign of severe vascular insufficiency Shooting lancinating pain or steady burning is the result of a traumatic ischemic, toxic or infectious neuritis This neuritis is often accompanied by a sensation of burning, aggravated by heat and relieved by cold Pain at rest however in the absence of intermittent claudication is not necessarily a symptom of vascular disease It may represent a multitude of neurologic and orthopedic lesions among which a lumbosacral arthritis an intervertebral disc tabes, peripheral neuritis pernicious anemia and diseases of muscles joints and tendons have to be considered

Pain on weight bearing is most likely a static difficulty due to weak arches pes cavus torn tendons corns calluses neuromata of the digital nerves or calcaneal spurs but it is certainly by itself not a vascular phenomenon although all of these irritative lesions may trigger vasospasm and may be present in addition to organic vascular disease

Night cramps in the calves may accompany calf muscle thrombosis but they are overwhelmingly due to increased irritability of the neuromuscular junctions or cord centers They may mean lack of sodium or calcium ions or a discharge of motor impulse from the cord often they are purely functional They are not indicative of vascular disease

Numbness and tingling of digits may or may not represent vascular disease They can be due to neuritides of all sorts but also to ischemia Radiculitis of the cervical and lumbar roots, due to compression by spurs or edema is a very frequent cause of these symptoms

What relieves the pain? Does standing still after exercise relieve it which is suggestive of circulatory impairment or does moving about help which may indicate neurogenic or arthrogenic pain? If heat improves the symptoms the pain may be due to vasospasm or a mild organic lesion since severe arterial occlusions stand heat poorly If cold relieves the symptoms the lesion may be inflammatory or due to a neuritis with antidromic vasodilatation (chapter 16) Elevation of the limb from the horizontal relieves the pain of venous obstruction with edema but also relieves that of inflammatory swelling as in a sprain a tenosynovitis or an abscess under the plantar fascia Hanging the feet over the edge of the bed or sitting up all night in a chair for the relief of pain is characteristic of arterial insufficiency This habit may pro-

duce a huge pitting edema which cannot be relieved by a horizontal position until the patient is heavily sedated or placed in an oscillating bed

Is there any edema? Is this swelling absent after a night's rest in bed and does it appear only in the course of the day? Or is it continuous? Does the swelling increase before menstruation? This is often true in chronic pelvic phlebitis, but also as a result of a hormonal effect on capillary permeability Is the swelling the result of a venous obstruction following childbirth or an operation? Did it appear soon after getting out of bed? Or has it always been there since childhood, the one limb being possibly larger than the other, and is there a cutaneous birthmark on this extremity or elsewhere on the body? Has there been any itching or scaling between the toes, followed by a red streak along the dorsum of the foot, and did the groin become tender or swollen? Was the swelling accompanied by pain, chills and fever, or was its onset slow, gradual and painless?

Does weather affect the symptoms? Is there a shooting pain from the back, aggravated by lying on a soft bed or by raising the leg? Does coughing or sneezing aggravate the pain? This is a root symptom and certainly not due to a vascular lesion Does the pain center in the popliteal fossa, and does this region seem swollen? Popliteal bursa often masquarades as vascular disease and may produce pressure symptoms on the popliteal nerve and vein. Is there swelling in several joints and is this intermittent? Does the pain center around an enlarged, big red toe which swells intermittently after dietary or alcoholic excess? Gout can be confused with vascular disease, all the more since at its acute onset an element of vasospasm may prevail, and since gout often accelerates arteriosclerosis

Many other questions may be asked and these are just samples of the questions frequently employed By careful questioning and proper interpretation of symptoms the examiner may have made important progress toward diagnosis

CHAPTER 6

THE EXAMINATION

THE METHODS OF EXAMINATION READILY FALL INTO TWO GROUPS To THE FIRST group of diagnostic procedures belong those which are available to everyone with limited equipment and which are sufficient to enable one to arrive at a clinical diagnosis The second group consists of equipment which is used for study of the disease, for research purposes and for advancing knowledge in this field They are by no means necessary for the efficient handling of the individual case but they lead to a better understanding of such lesions and require a group study by several workers In this chapter only the methods that have been used by our group will be discussed and no attempt will be made to give a complete list of diagnostic procedures

1 EVALUATION OF LOCAL CIRCULATORY STATUS

The patient must be completely undressed and lie flat on his back in a room whose temperature varies preferably between 70 and 76 F This, of course is not always possible in hot weather The use of rooms with con trolled temperature and humidity is desirable for purposes of controlled re search but is not necessary for clinical study Daylight is preferable but good artificial light is satisfactory

Inspection of the affected area may give a great deal of information The heart may show increased pulsation with the apex beating outside the mid clavicular line The intercostal spaces to the right or left of the sternum may show a pulsating bulge or the jugular fossa may be filled with a mass pressing on the trachea and producing dyspnea or stridor The abdomen may show an area of pulsation to the left of the umbilicus or there may be a visible mass in the flanks One arm or leg may show pallor compared with its fellow or one limb may be smaller the muscles being atrophic in the calf or in the thigh in the forearm or upper arm The skin may be atrophic shiny and glossy it may be ulcerated or can be frankly gangrenous The area of gan grene may be dry the line of demarcation being very definite and showing no inflammation around it or it may be swollen and purulent, with a zone of reactive hyperemia, but with insufficient demarcation The veins may be distended, even in the horizontal position, or collapsed They may show a pattern characteristic of long or short saphenous involvement or can exhibit a course typical of collateral circulation which follows deep venous obstruc tion (fig. 18) The skin may be indurated, with reddish brown pigmentation



FIG 18 Pattern of collateral circulation in deep venous insufficiency Note the supracondylar and subtrochanteric fat pads due to pluriglandular insufficiency

The induration may contain an ulcer in its center The ulcer may have healthy granulations and a sharp edge, it may show pale atonic granulations or areas of slough, with yellow or green exudate, the margins can be undermined (fig 19) The veins may show segmental, sharply delineated red cordlike indurations, or small hard nodules The lymphatics may exhibit red streaks running cephalad, and the regional glands can be involved One extremity may be visibly more swollen than the other

If now the patient sits up and hangs his feet down, or stands up if he can, the postural change may bring about a modification of the picture The pallor (of the lower extremities) may give way to a marked rubor, the upper edge of which can be noted Previously collapsed veins may stand out showing marked back pressure An inflamed nodule or a low-grade periphlebitis will now become more obvious Should the patient be unable to sit or stand, a blood pressure cuff pumped up to 60 mm. of mercury over the thigh will

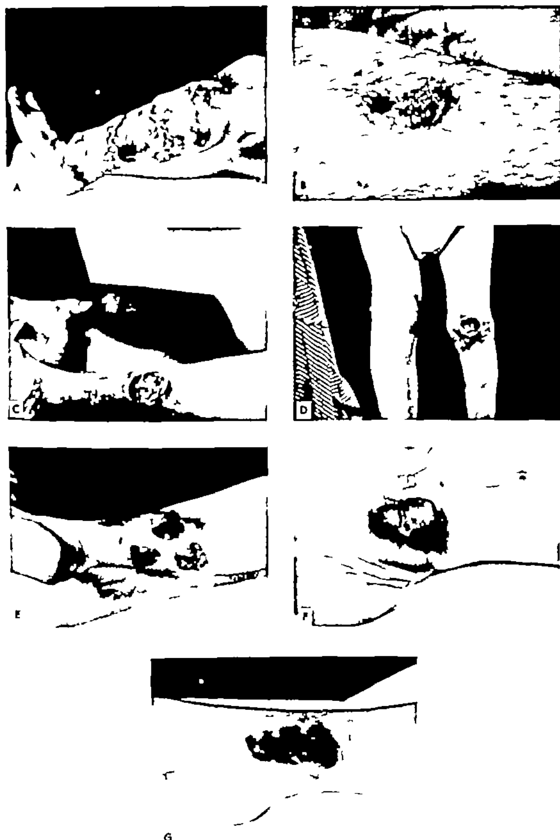


FIG 19 Chronic leg ulcers of various etiology (A) Postphlebitic ulcers, riding over incompetent perforators. (B) Arteriosclerotic ulcer initiated by trauma. (C) Punched out luetic ulcer with necrotic base. (D) Painless trophic ulcer in syringomyelia. (E) Multiple shallow ulcers in a diabetic, necrobiosis lipoidica the reddish papules broke down, became infected and formed apple-jelly colored plaques. (F) Ulcerating melanoma. (G) Hypergranulating persistent ulcer terminating in fulminating lupus erythematosus.

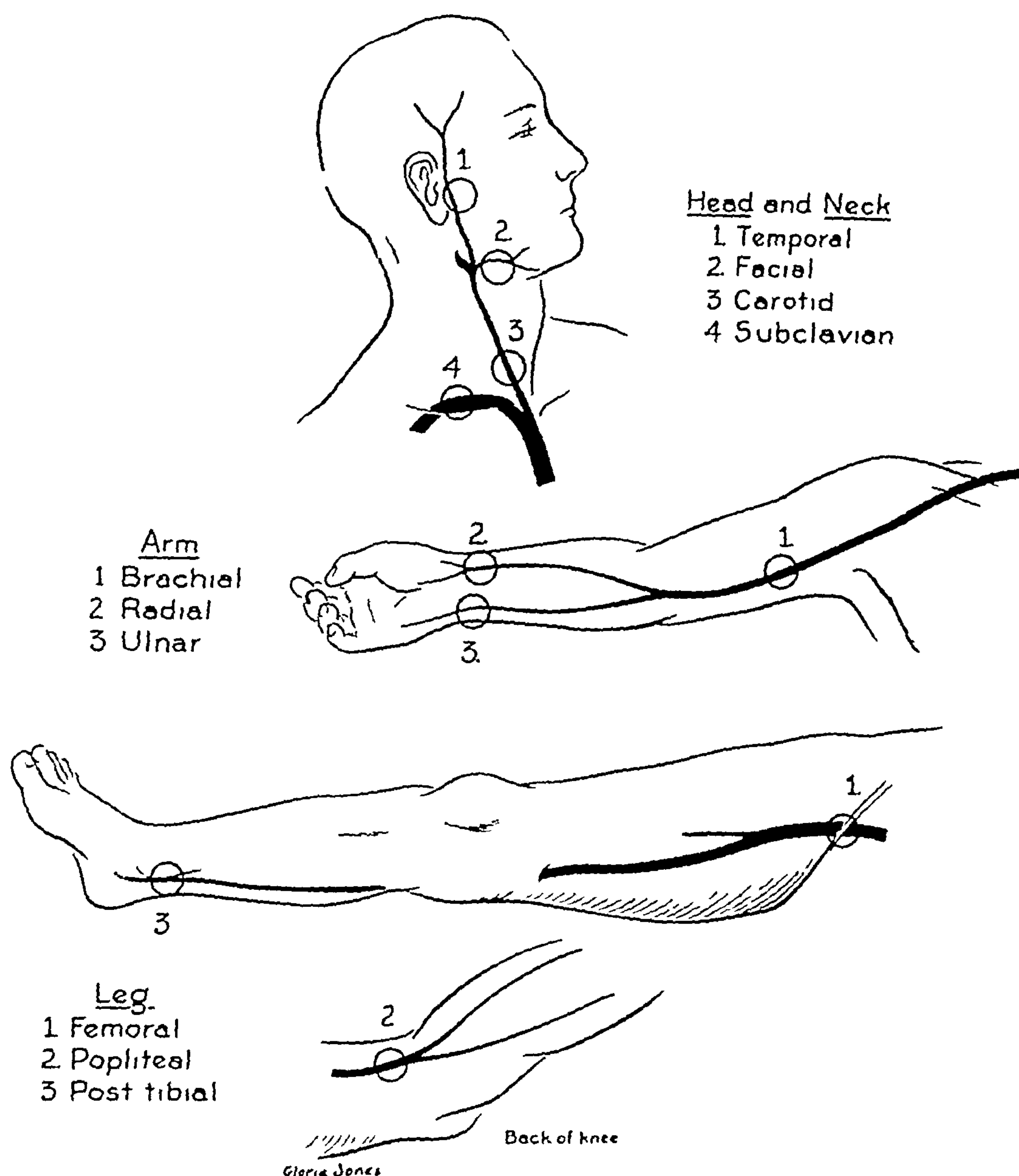


FIG 20 Palpation of pulses at typical locations In addition to the rate, one can establish the presence of a full, a diminished or an absent pulse, one can also feel a soft compressible or a hard, nodular, rigid artery A medial calcification feels like a goose neck Absence of pulse does not rule out patency, there may be nonpulsatile flow in the artery

produce the same effect The rise in venous pressure may bring out the symptom of erythromelalgia The patient should now be turned on his abdomen, or at least on his side, so that the popliteal fossa can be inspected This is a small but significant area and any swelling, discoloration or unusual pulsation should be carefully noted

Palpation in case of vascular disease is primarily directed toward the presence of normal or abnormal *pulsation* The apex of the heart, the entire cardiac area, the jugular fossa, the common carotid, the axillary, brachial and radial arteries, the abdominal aorta, and the femoral, popliteal, posterior tibial and dorsalis pedis arteries must be palpated (fig 20) In all these locations one looks for the presence, absence or diminution of pulsation. Diminu-

tion of pulsation can be determined by comparing it with the opposite side if that side is unaffected One also notes the presence of a thrill on palpation which may denote an aneurysm a narrowing of the artery or a large plaque producing whirls and eddies in the blood stream

Some arteries such as the subclavian the ulnar or the digital arteries are seldom, if ever accessible to direct palpation Indirect methods for determining their patency will be described elsewhere Also the dorsalis pedis artery may be absent because of a high division of the anterior tibial artery or it may be found more laterally than usual in a certain number of cases

Palpation of peripheral arteries also reveals small or large pulse pressures irregular beats pulse deficits and the thickening of the arterial wall its tortuosity or nodular structure Palpation of veins may detect thickening, calcified phleboliths elicit pain and detect differences in skin temperature Deep pressure on flexor muscles of the calf which produces pain may lead to a suspicion of thrombosis Pain from pressure on the calves may be produced by a blood pressure cuff normally pressures up to 180 mm of mercury are tolerated without much discomfort [3]

Palpation with the volar surface of the three middle fingers of the examiner s hand or with the back of the hand will detect differences in *skin temperature* as little as 1 degree F or ½ degree C being detectable If the room is cool enough (about 70 F) a small difference in skin temperature between the two big toes, or between that of fingers of the same hand is significant The warmer the room the more difficult it becomes to observe such changes However in a warm room or after block of the regional sympathetics, a difference noted in temperature is all the more significant

The palpating hand can demonstrate a soft pitting edema and detect a hard nonpitting lymphatic type of obstruction The palpating hand can delineate the hard, scalloped margin of a thrombophlebitic induration, the presence of hard fixed or discrete lymph nodes and the presence of masses in the extremities or at their roots which exert pressure on or invade the lymphatic trunks

Palpation detects the presence of abnormal sweating, generalized or localized (*hyperhidrosis*) This sweating may be limited to a single extremity or even to the distribution of a peripheral nerve Or it may appear at the border of a dry nonsweating area such as a marginal hyperhidrosis seen in peripheral nerve lesions or after sympathectomy The extremity may be excessively dry (*anhidrosis*) owing to interference with the blood supply of the sweat glands but more commonly to a lack of nerve conduction which leads to a block of the sweat fibers A complete lesion of a peripheral nerve can be roughly mapped out by feeling for the dry area so also can the extent of a sympathectomy be determined since the sympathetic fibers carry the sweat fibers There is however a possibility that sudomotor activity and vasomotor activity do not completely overlap In order to bring out sweating and nonsweating areas more distinctly the patient can be given hot tea and 15 gr of aspirin and a large heat cradle placed over him While more accurate tests for sweating are available and will be described much can be

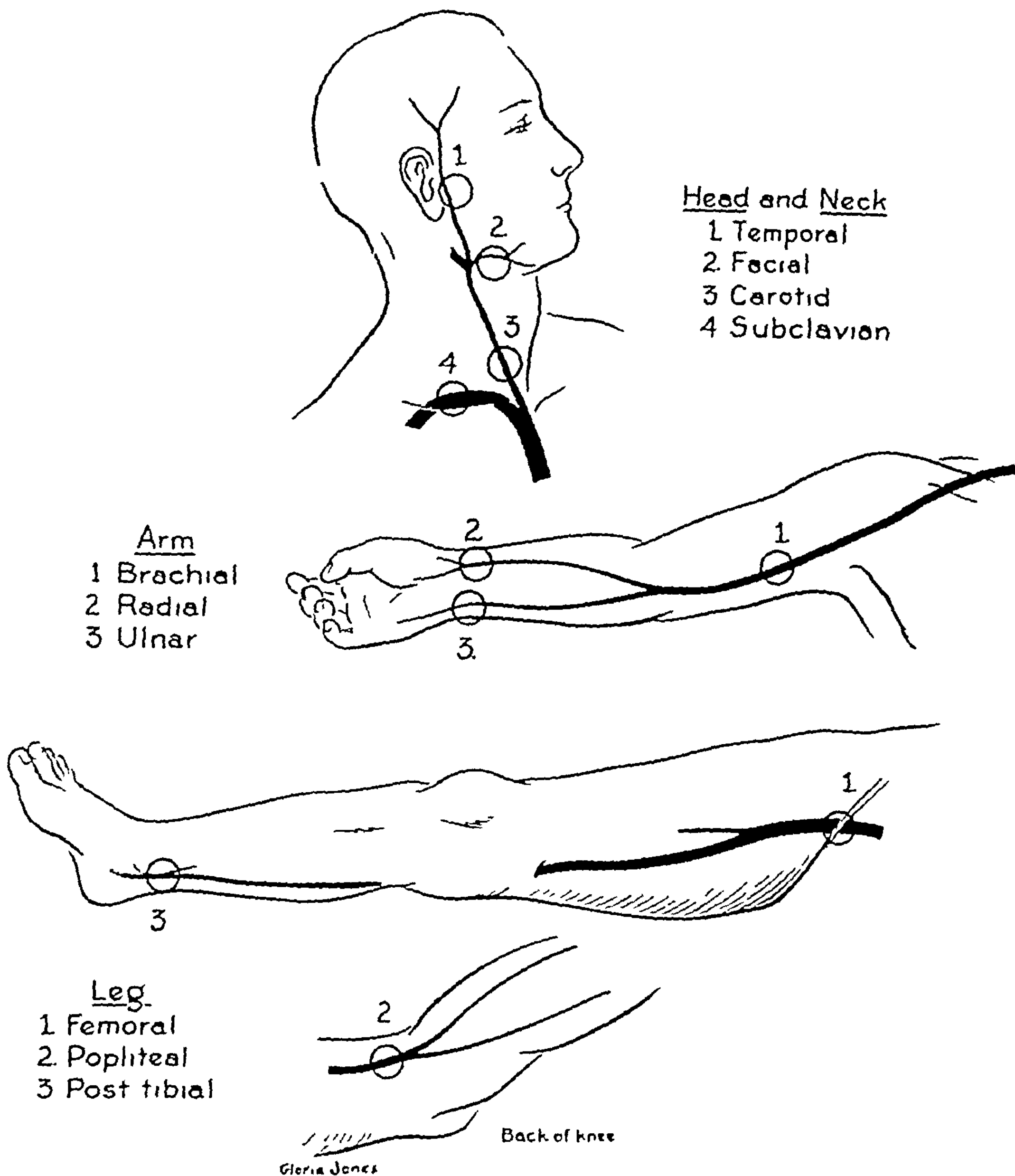


FIG 20 Palpation of pulses at typical locations In addition to the rate, one can establish the presence of a full, a diminished or an absent pulse, one can also feel a soft compressible or a hard, nodular, rigid artery A medial calcification feels like a goose neck Absence of pulse does not rule out patency, there may be nonpulsatile flow in the artery

produce the same effect The rise in venous pressure may bring out the symptom of erythromelalgia The patient should now be turned on his abdomen, or at least on his side, so that the popliteal fossa can be inspected This is a small but significant area and any swelling, discoloration or unusual pulsation should be carefully noted

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learned by simple palpation With the help of a magnifying glass, drops of sweat are readily visualized Certainly the extent of a sympathectomy can roughly be determined in this way

Blood Pressure

Blood pressure must be taken on both arms, using the first sound for the systolic and the disappearance of the sound for the diastolic pressure If the lower extremities are affected or if a coarctation of the aorta is suspected following the general physical examination, blood pressures are taken on both lower extremities The cuff is placed on the thigh and the stethoscope over the popliteal fossa with the patient in the prone position, in thin individuals both systolic and diastolic pressures can be obtained If this is impossible, the appearance of a palpable pulse is taken as the systolic pressure when the cuff is slowly deflated, the diastolic pressure cannot be determined

With the advent of the Pachon oscillometer, accurate readings of blood-pressure could be obtained on the lower extremity with a double cuff The absence of blood pressure, or a difference of blood pressures amounting to more than 15 mm of mercury between the two sides, is of course significant Systemic high blood pressure calls for a detailed examination directed toward operability

Detailed description of the fundamentals of venous pressure may be found in the excellent monograph of Burch [5]

Venous Pressure

Internists use determinations of venous pressure to detect early heart failure of the back pressure type For the vascular surgeon, the measurement of venous pressure is useful in the diagnosis of *cardiac tamponade.* When the pericardium fills with blood or exudate, venous pressure rises, arterial pressure falls and pulse pressure is small Clinically this is detectable by the distention of the jugular veins When venous pressure gradually falls after previously being elevated and arterial pressure does not rise, heart failure is increasing In *pulmonary embolism* the obstruction of the pulmonary artery dilates the right side of the heart and this increases the back pressure in the superior and inferior vena cava. In *peripheral venous obstructions,* the venous pressure rises and this may be accentuated by muscular exercise (fig 21) Venous pressure may increase generally on exercising a localized area, such as the two legs

When the patient is in the horizontal position and the vein to be studied is roughly at a level with the right auricle, there should be little distention In raising the arm above this level and noting the height at which the vein collapses, one can express venous pressure in centimeters of water. This is particularly useful when the other extremity can be used for control, as, for instance, in thrombosis of the axillary vein or in an iliofemoral venous thrombosis However, a simple apparatus based on the U tube principle permits an

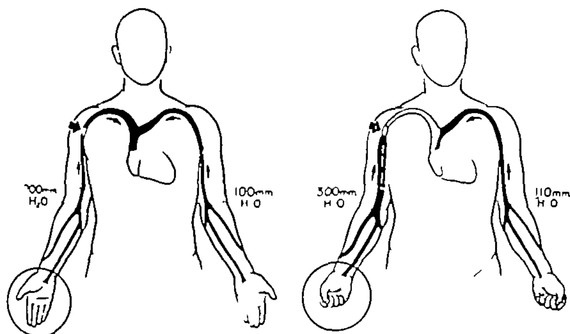


FIG 21 In the case of an axillary venous thrombosis (right) the resting venous pressure in the right cubital vein determined at heart level is higher than that in the left However after vigorous flexion and extension of both elbows, the venous pressure in the right arm rises so visibly and palpably that it need not be measured On the unobstructed left side there is a slight rise from 100 to 110 mm. Hg on the obstructed side the rise is from 200 to 300 mm. Hg. A true claudication of the arm develops with pain and fatigue. (Burch G E. Jr A Primer of Venous Pressure Lea and Febiger Philadelphia, 1950)

accurate estimation of venous pressure we reported on this in 1929 [4] Spinal manometers may be used Our more recent experience has been with the Burch apparatus the Phlebaumanometer[5] (fig. 22) Venous pressures may also be simply estimated by palpation in the lower extremities before and after exercise Deep venous obstruction and deep venous insufficiency can be differentiated (chapter 13)

The Oscillometric Curve

No peripheral vascular disease can be thoroughly studied without an oscillometer and since it is readily available and inexpensive its routine use is strongly recommended There are several instruments on the market and one should preferably be employed which is light easy to repair and portable (fig. 23)

The apparatus consists of a sensitive aneroid which registers the pulsation of a cross section of the limb. When the cuff on the limb is a single one it will bear the impact of the incoming pulse wave the pressures in millimeters of mercury are registered on one dial, whereas the other dial is reserved for the swing of a needle measured in centimeters The swing at first is very small, then increases with the lowering of the pressure in the cuff reaches a peak and then decreases again until it registers less than a one half centimeter swing Such a normal oscillometric curve obtained with a single cuff is shown in curve 1 figure 24 It should be emphatically stated that this

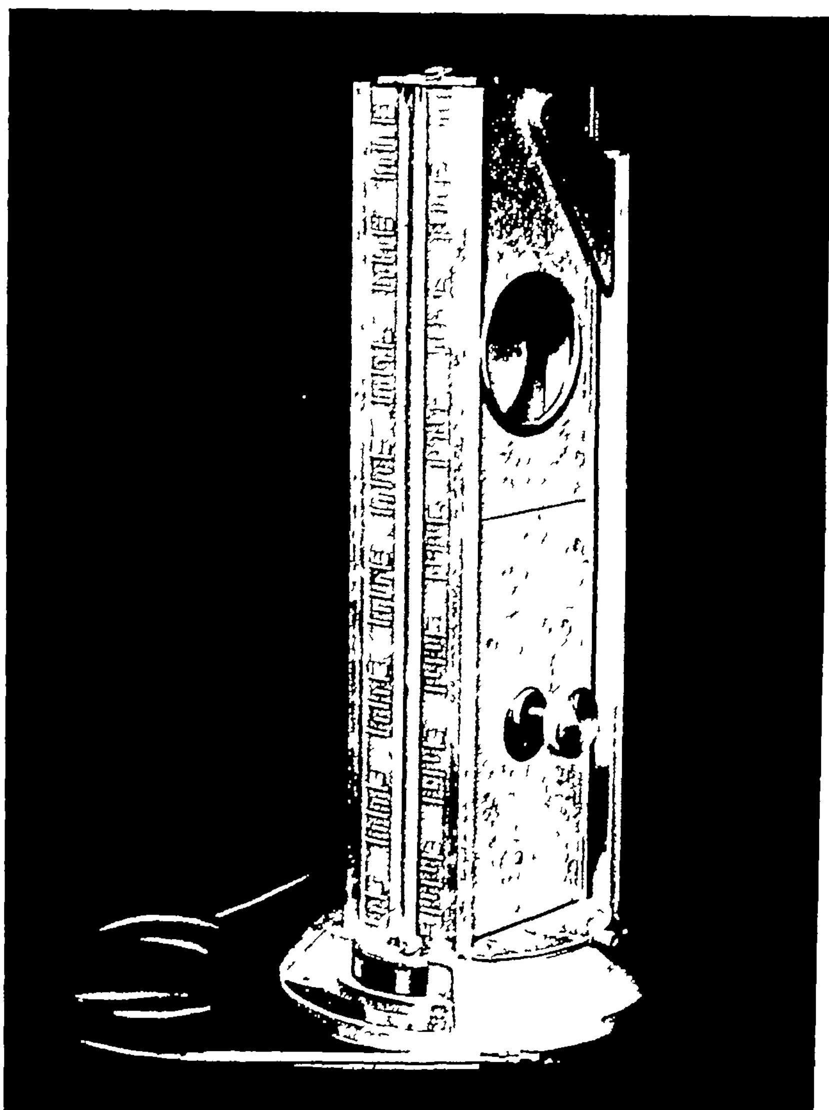


FIG 22 Phlebaumanometer (W A Baum Co, Inc)

does not measure the blood pressure in the extremity The initial swing begins at a higher level, the peak is between the systolic and diastolic pressures (the mean pressure of Vaquez), and the diastolic pressure can not be determined accurately by this curve The graph simply represents the transmitted pulsation of a certain cross section of an extremity Its shape will depend on the stroke volume of the heart, on the patency and size of the major arterial pathways, on the elasticity of the arteries, on the size and consistency of the soft tissues of the limb and on terminal, arteriolar or venocapillary resistance Thus, if an oscillometric curve is obtained from symmetrical areas of two extremities, the following characteristics of the curves should be looked for

Curve 2 The needle begins to swing at a lower mercurial pressure and reaches a smaller peak, but its downward curve is identical with the normal This means an arterial narrowing or obstruction *proximal to or at the site of the cuff*

Curve 3 The needle begins to swing at the same mercurial level, but

instead of the curve describing a sharp peak it flattens out to a plateau which is at a lower level than the peak but which extends over several determinations taken at intervals of 10 mm of mercury Its downward swing again has not changed very much if at all Such a curve is obtained in the presence of inelastic arteries which do not transmit the pulse wave with the normal intensity if there is a great deal of indurated soft tissue, as in a chronic phlebitic or lymphatic edema the same type of curve may be obtained

Curve 4 The pulse waves start at a lower level but end sooner there is a flattening of the entire curve and the peak is lower This is a generalized increase in vasomotor tone such as one sees in a position of erect posture as compared to the horizontal position

Curve 5 The curve is very low or completely flat indicating no pulsation which the apparatus can pick up This of course is partly due to the limitations of the instrument since a sensitive finger plethysmograph or an electronic oscillometer may register a pulse wave when the oscillometer at the wrist is silent However in general this curve simply means that none of the larger arteries are pulsatile although the limb may still be amply nourished by nonpulsatile collateral circulation

Curve 6 The pulse waves come in at a higher level the peak is just as high as on the unaffected side but occurs at a higher pressure and the downward curve is seen at a higher mercurial pressure The entire curve has *shifted to the left* This means an increased terminal resistance which may be

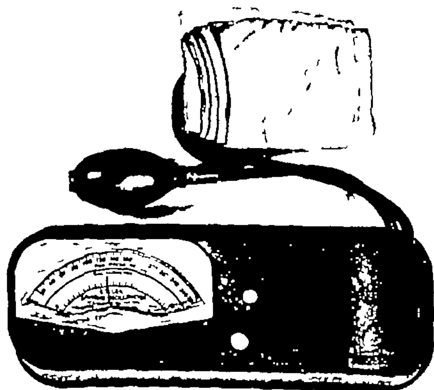


FIG 23 Oscillometer (Collens-Wilensky)

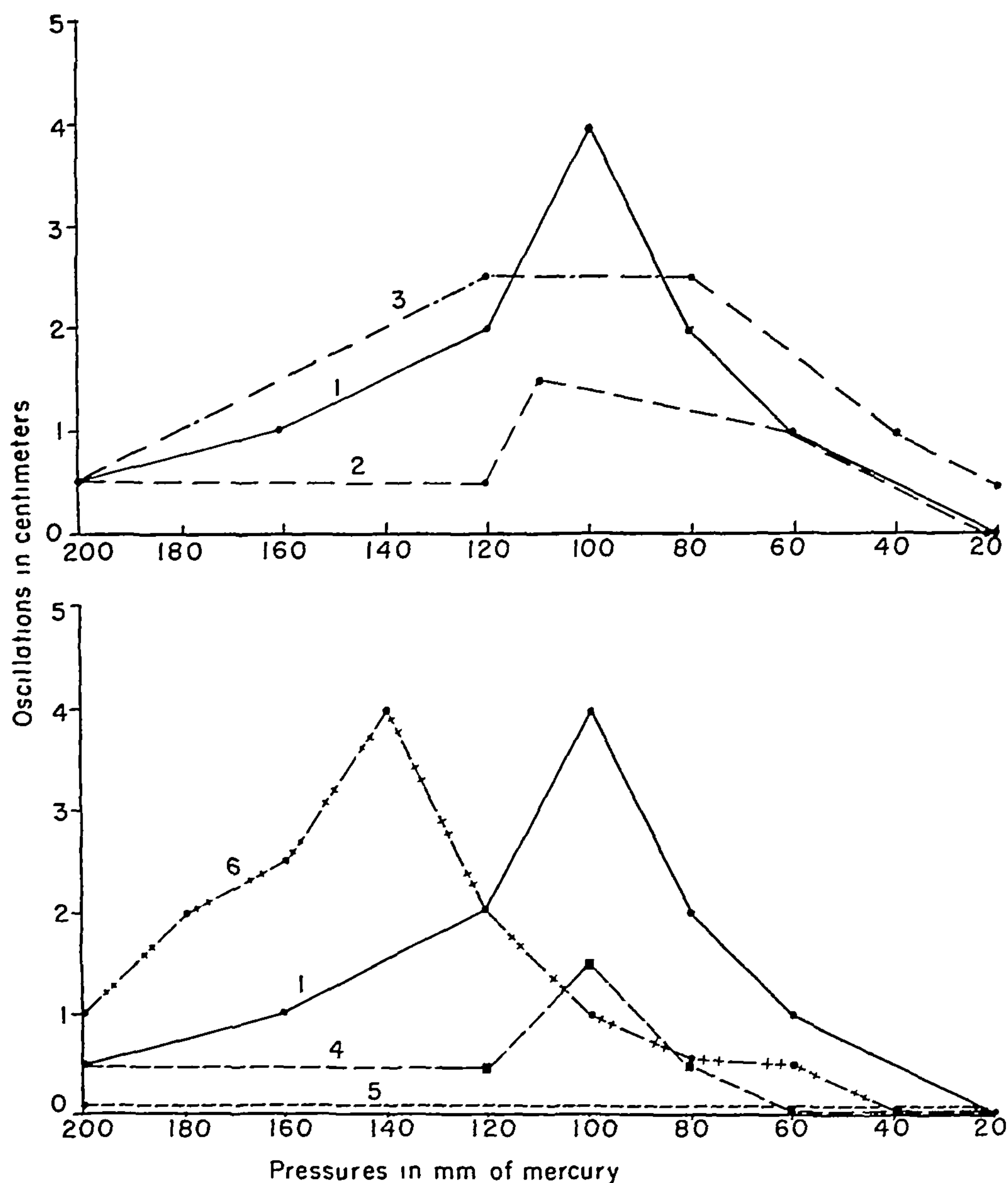


FIG 24 (1) Normal oscillometric curve The index is 4/100 mm Hg but the shape of the curve is much more revealing The rise and fall of the curve indicates distensibility of the main vessels and the cross section of the extremity at the level of the cuff (2) A flat curve with a small peak and a gradual downward slope Obstruction is proximal to or at the site of the cuff (3) The ascent of the curve is normal, but flattens to a plateau, the downward swing is normal This is a poorly transmitted pulse wave because the vascular tree or the surrounding tissues have lost their distensibility. (4) The curve is flat down to 120 mm Hg as in curve 2, then shows a slight rise and fall This is a mirror image of curve 1, the normal curve, and is due to an increase in vasomotor tone such as one sees in the erect posture (5) The curve is absolutely flat, the needle does not swing, indicating no pulsation that the instrument can pick up This means lack of pulsatile circulation (6) The pulse waves come in at a higher level, the peak is reached at higher pressure The index is 4/140 instead of the index of 4/100 shown in curve 1 The entire curve has shifted to the left This means an increased peripheral resistance as seen in diastolic hypertension Venous stasis causing increased capillary pressure may produce it.

The patterns represent trends and are not curves obtained from individual patients They must be interpreted in conjunction with the clinical findings

organic or spastic. Venous stasis producing increased capillary pressure may produce it. Diastolic hypertension may be present on erect posture.

The oscillometric curve may be smaller and flatter but, most important, the swing of the needle is slow and wormlike. The pulse wave here is due to a collateral circulation partly compensating for a segmental obstruction of a major artery. Following restoration of continuity of the major pathway, the needle swings more widely and much faster (increased crest time of the plethysmograph).

A fairly normal oscillometric curve may become flatter and shorter following a short intensive muscular exercise, presumably because the stenotic arterial segment cannot deliver enough blood to the level of the cuff; the blood is diverted to the active muscles. This decrease in oscillometric curve also occurs in the sympathectomized limb, and one has no evidence of angiospasm. Arterial stenosis can be demonstrated by angiogram (fig. 25).

An oscillometer equipped with a double cuff does not register an oscillometric curve, but is useful for taking blood pressures on upper and lower extremities; these are independent of any variations of hearing since a steth

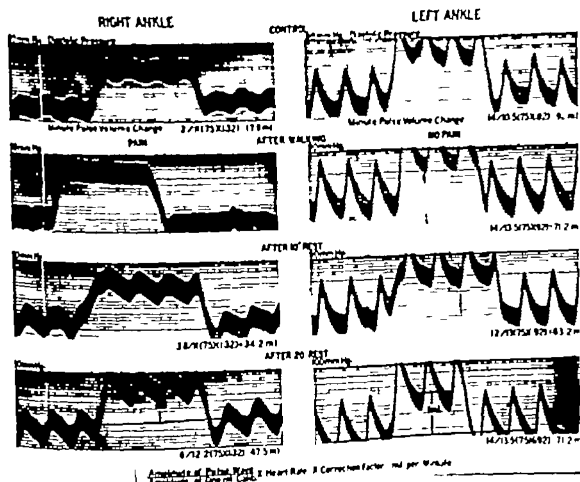


FIG. 25. Venous occlusion plethysmograph. This patient had intermittent claudication in the right calf and at both ankles, and an occlusive pressure of 60 mm. Hg at thigh level. Note the small pulse wave at the right ankle which disappears on walking and increases over the pre-exercise level after 20 minutes of rest. Note the flattened crests on the right compared to the sharp, high crests on the opposite normal side. (Courtesy of Dr. Carl Johnson.)

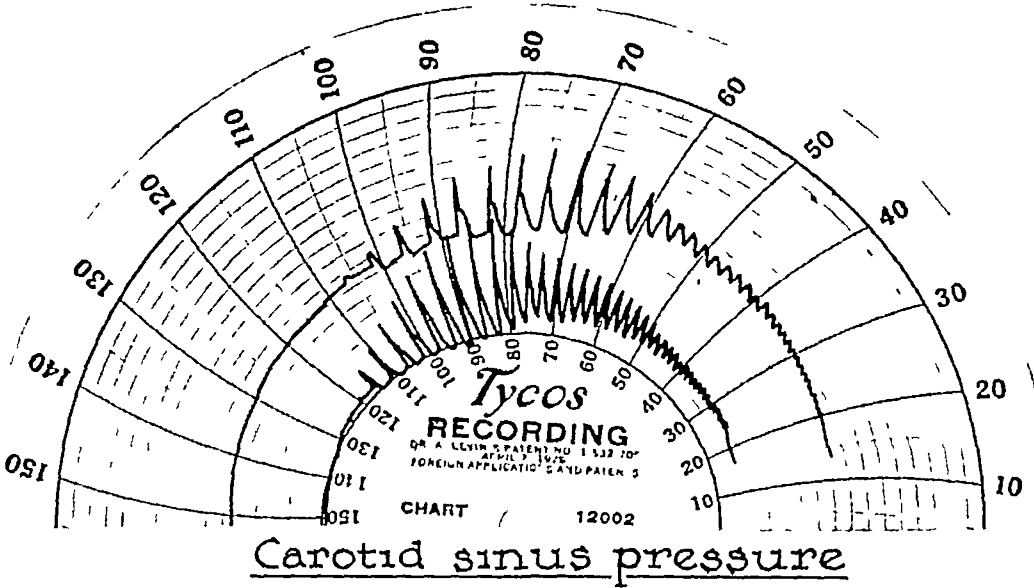


FIG 26 Double-cuff oscillometric curve before and during pressure on the left carotid sinus Note the fall in pressure, the bradycardia and the decrease in the height of the individual pulse waves in the upper curve, taken during pressure on the carotid bulb

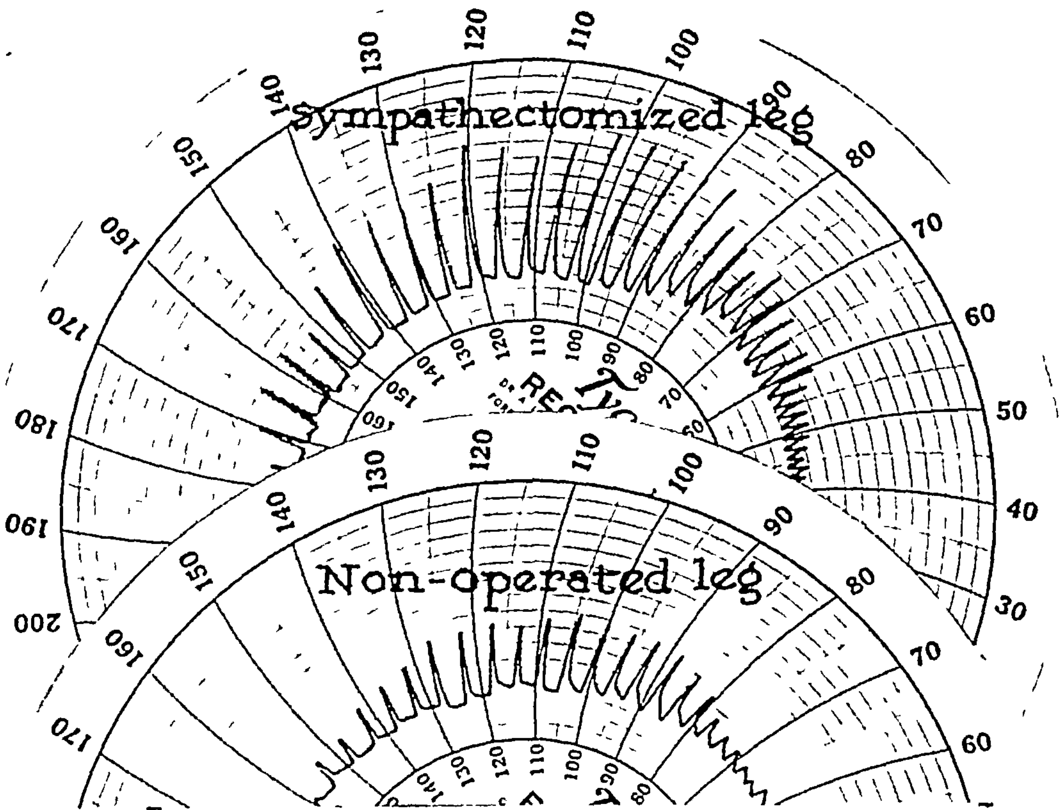


FIG 27 Single-cuff oscillometric curves of a student nurse suffering from hyperhidrosis of both lower extremities five days after one side had been operated upon There is no vascular disease and hence the response on the pulse wave is marked Regain of vascular tone has not yet taken place 10 years later the patient was still free of hyperhidrosis

oscope is not used In figure 26 note the fall in blood pressure from 120/75 to 100/60 mm Hg together with bradycardia and decrease in stroke volume when unilateral carotid sinus pressure is exerted The systolic pressure is read when the first swing of the needle is noted and this is often 5 to 10 mm of mercury higher than that obtained by auscultation The diastolic pressure is read when the larger swings suddenly decrease to three even beats after the base line of the pulse waves has risen In the past we made extensive use of such a self registering oscillometer which produced a permanent record but which is not necessary for clinical study (fig 27) A single and double cuff arrangement was available None of these oscillometers nor the many plethysmographs, can measure actual flood flow although many calculations have been made

Skin Temperatures

The temperature of the skin in various parts of the body depends on a number of factors The temperature of the room the amount of perspiration food intake, smoking, fever basal metabolism the emotional state and the position of the patient are some of the better understood influences acting on skin temperature Therefore a basal state is desirable As far as a room with controlled temperature and humidity is concerned this is available in teaching and research institutions but unnecessary and unavailable for most of the clinical studies The constant temperature room will be discussed on page 98

Without controlling these factors a single determination of the skin temperature of a digit is meaningless However determinations are valuable when they are taken *from symmetrical areas of the two extremities before and after vasoconstrictor tonus is released, or when there is a sudden drop of skin temperature at a certain level of an extremity* Under such conditions, all other factors are equal and therefore the figures have relative value

Simple methods in use to determine the differences in temperature are as follows

(1) *Palpation of the skin* with the tips of the three middle fingers is a rough but useful estimate of differences in skin temperature With some practice differences as little as 1 degree F (½ C) are detectable In a cool room with a temperature of 65° F (18 C) such differences are more obvious than in a warm room of 75° F (25° C) In a hot room with temperatures of 88° to 90° F (31 to 32 C) vasomotor tone is abolished and thus any organic lesion would manifest itself by a greater difference in temperatures Fortunately humidity of the surrounding air seems to have little influence on the temperature of the skin[6] Surrounding a cool foot with a thick layer of absorbent cotton wrapped from toes to knee may elevate the skin temperature to a level of normal vasodilation (31 C)

A palpable drop of skin temperature between different digits of the same extremity or of the same digits on symmetrical areas of the contralateral extremity means decrease of blood flow but whether this is spastic or organic

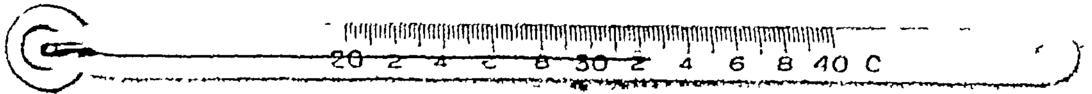


FIG 28 Mercury skin thermometer registering in centigrades (Rauscher and Betzold, Chicago)

or a combination of both is thereby not decided A sudden drop in the temperature of a leg or arm after it has been exposed to room temperature for 20 to 30 minutes is a good enough estimate of circulatory impairment Should this be on a spastic basis, the measures described to eliminate vasomotor tone (p 67) will abolish this difference A rise of skin temperature after paravertebral block, which reaches or even exceeds the palpable temperatures of the opposite digit, denotes that vasomotor tone was a factor in producing the difference in temperatures Since sweating permits cooling of the skin by evaporation, a moist skin may feel cooler than a dry one This is the main reason why determination of skin temperatures by thermometers is preferable

(2) *The mercury skin thermometer*, a small, inexpensive skin thermometer, has been in use in our clinics for many years (fig 28) It is true that the temperature readings vary according to the pressure exerted on the skin by the bulb, also, less than ½ degree C differences are within the limits of error In addition, the thermometer is slow in its response and may take two minutes to come to equilibrium Nevertheless, one can derive considerable information from it, and although it is very fragile it is easily replaceable The thermometer is calibrated in Centigrade or Fahrenheit It can be put into a pocket

(3) *The thermocouple galvanometer* is based on the principle that if one thermocouple is maintained at a constant temperature, such as by ice water or warm water of known temperature, the other thermocouple is connected with the skin with the temperature to be determined A current then flows over the connecting wire causing deflation of a string galvanometer which is calibrated in centigrades or Fahrenheit These instruments are fragile, cumbersome and not very portable Huge stationary galvanometers can, of course, be built in connection with rooms of controlled temperature and humidity in research institutions

(4) *The electric thermometer* (fig 29), built on the principle of a Wheatstone bridge, operates with removable and replaceable dry batteries Its scale is calibrated in Fahrenheit It is portable, reads quickly and flawlessly, with an error of 0 5° F or less It has been constructed for us by the General Electric Company years ago and has been on heavy duty since At present a McKesson instrument is in use. It is not absolutely necessary for a clinical study but has been in frequent use for purposes of investigation, or to check on the accuracy of the palpatory findings and the mercury thermometers.

For interpreting these readings it must be clear that all skin temper-

atures, even under controlled conditions only reflect the state of the cutaneous circulation not that of the deeper structures such as muscles Not only are measures known which raise skin temperatures without affecting the temperature of muscle but there are procedures which are said to produce a fall in deep temperatures when surface temperature rises [7] This has been one of the criticisms leveled against the use of sympathetic block temporary or permanent in the treatment of peripheral vascular disease These findings may require reinterpretation on the basis of Barcroft s hypothesis of a double circulation in muscle anastomotic and nutritive [8] If reflex heating and sympathetic block open the arteriovenous shunts neither the muscle temperature will rise nor will sodium clearance methods show any increase in circulation This subject has been discussed in chapter 3 Vasomotor Apparatus

Under conditions of daily life in the upright position or after exercise, the relation of skin to muscle temperatures has not been sufficiently investigated Clinically a warm skin in obliterative vascular disease is certainly a better prognostic sign than cold skin and denotes in the absence of pulsatile arteries a fairly well maintained collateral blood supply Patients with a high vascular tone will exhibit lower skin temperatures exposed to customary room temperatures than those with a lower tone Basal metabolism is an important factor

The great disadvantage of skin temperatures for measuring blood flow lies in the large thermal lag involved so that it will not pick up suddenly even

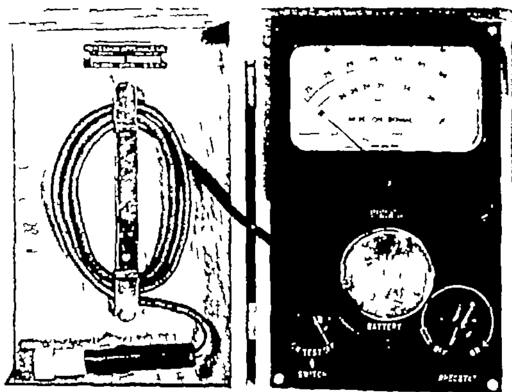


FIG 29 McKesson s electric thermometer A rugged instrument, this is suitable for clinical use on the wards and in out-patient departments. It is light and portable.

if drastic changes of circulation occur [9] Nevertheless, as recently pointed out by Catchpole and Simeone,[10] it is still one of the simplest methods to follow changes in circulation and an indirect method of estimating blood flow. Of all the methods used to determine blood flow, the study of skin temperatures, with obvious limitations, is the most practical one, especially if environmental temperature is controlled

The Histamine Flare

Even although the "triple response" of Lewis to a cutaneous puncture of histamine[11] has been repeatedly described, there is still some confusion as to the clinical significance and value of this test When 1 1000 histamine acid phosphate is pricked or, preferably, as we have suggested,[12] intradermally injected into the skin, a wheal, a small purple spot and a flare develop Neither the purple spot, which is due to local capillary stasis, nor the wheal, which is due to increased capillary permeability, are of any interest from the standpoint of circulatory efficiency The size and intensity of the flare is what we look for This is due to an axon reflex, which originates from stimulation of the cutaneous sensory network by passing of the impulse to a division of the afferent fiber and by an efferent impulse back to the skin producing vasodilatation and itching Such a flare can be produced by substances other than histamine, such as morphine or codeine, or by freezing, burning or even scratching the skin By scratching the skin from thigh to ankle with a fork, one can observe the absent red line at the level of the insufficient blood supply This can be well demonstrated in a warm class room. It is an important defense mechanism of the skin against injury, since it evokes a hyperemia at the site of trauma For practical purposes it is enough to examine the site of histamine punctures at the end of five minutes and grade the flares on the basis of three to zero Three is a normal, full flare, two is a diminished, one is a minimal and zero is an absent flare Placed on various levels of an extremity this is, with some reservations, our best method of determining the proper level of amputation In the presence of vasospasticity the test should be repeated under paravertebral block or spinal anesthesia It can be done in the operating room after spinal anesthesia has been induced prior to amputation

The negative flare means that cutaneous circulation is inadequate to produce the reflex hyperemia Since this axon reflex is dependent on an intact sensory nerve supply of the skin, the flare may also be absent in the presence of normal circulation when there is a degeneration of cutaneous nerves Thus any lesion or block proximal to the posterior root ganglion leaves the histamine flare intact, this is true of spinal anesthesia or of a cord lesion producing anesthesia But when the peripheral nerve is affected by trauma or compression, and enough time has elapsed for degeneration, the flare does not appear. In a fresh transection of a nerve, the flare will persist for several days, until peripheral degeneration is complete Conversely, when regenera-

tion occurs, histamine flares reappear Peripheral nerve lesions can be thus mapped out with the help of histamine flares

Since the intradermal injection of histamine evokes an almost instanta neous sensation of burning pain the absence of this pain reaction is good evidence of absent afferent conduction When the burning pain appears not after a second or two but 20 to 30 seconds later one can assume that the rapidly conducting myelinated fibers are nonfunctioning and only the slowly conducting nonmyelinated fibers are active This is often the case in tabes or in diabetic neuropathy [13] Thus in diabetic vascular lesions the histamine response may give information regarding the vascularity of the limb and also the integrity of its nerve supply which may be completely lost together with the tendon reflexes or may be selectively intact The loss of myelinated fibers and the persistence of nonmyelinated fibers give rise to peculiar vaso-motor phenomena observed in tabes and in diabetes but also in nondia betic ischemia of peripheral nerves which are not unlike those observed in causalgic states

The Venous Filling Time

Collens and Wilensky[14] described a simple test designed to study the rapidity with which veins previously collapsed fill up through the arterial tree They empty the veins of the foot by elevation and then allow the limb to hang down The dorsal veins of the foot should fill normally within 5 to 10 seconds when there is marked impairment of arterial inflow from 60 to 180 seconds may elapse before the veins fill The objection to this test is that with defective venous valves the veins may fill retrograde from above instead of through the capillaries For this reason a simple modification has been adopted and used for many years The limb is elevated and the venous blood allowed to drain from the extremity With a blood pressure cuff around the thigh, 60 mm pressure are rapidly developed, this does not obstruct arterial inflow but throttles venous outflow Within 5 to 10 seconds the veins on the dorsum of the foot stand out There may be some difficulty in reading the end point, that is the filling time but when the vein is well raised above the skin level or if venous pulsation becomes visible the elapsed time is noted be tween the inflation of the cuff and this point. Increased vascular tone either because of sympathetic activity or local chilling, may greatly delay venous filling time and therefore it is wise to perform this test in a warm room or after the limb has been wrapped in a warm moist towel

The test is most useful for determining improvement or deterioration of circulation after certain therapeutic procedures A venous filling time of more than 10 to 15 seconds is certainly pathologic often one extremity shows a venous filling time of 60 seconds against 180 seconds of the contralateral extremity although both are pulseless and show no difference in skin tem-perature The test is a fairly good guide in timing the exposure of films in femoral arteriograms In the absence of palpable pulses, it measures the efficiency of collateral circulation The extremity with the filling time of

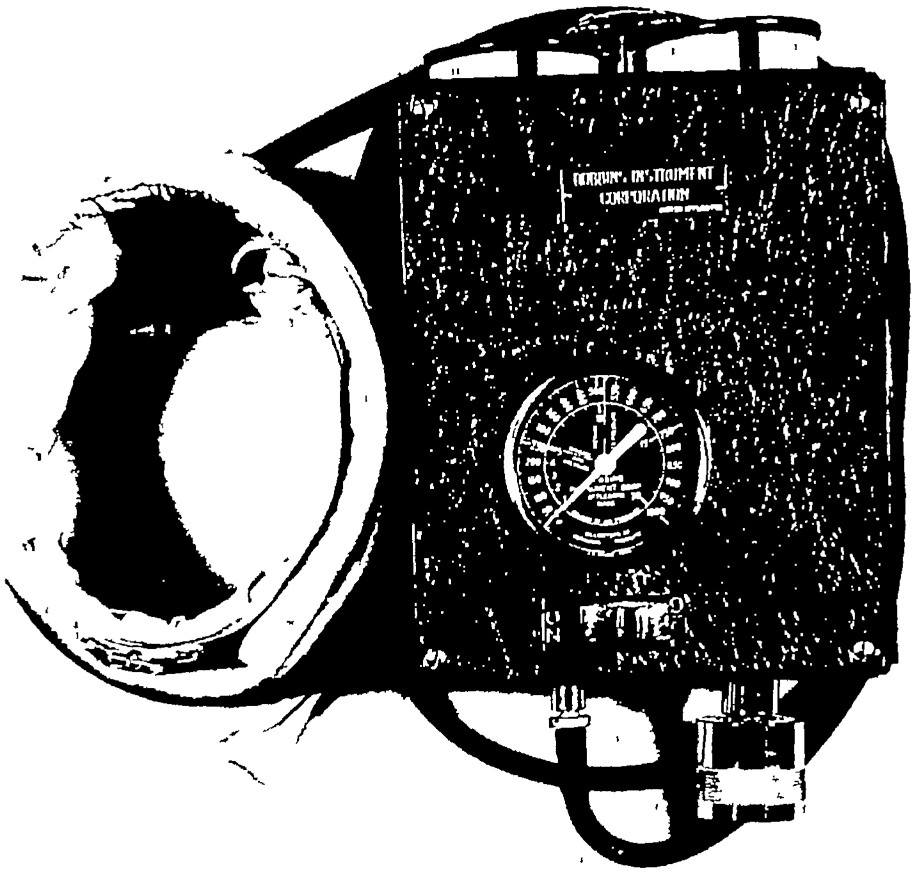


FIG 30 Inflation of the cuff is almost instantaneous and is accomplished by a freon element For the upper extremity, 300 mm Hg pressure is necessary to produce complete ischemia, for the lower extremity, 400 to 500 mm Hg Inflation and deflation are accomplished by a switch placed below the dial (Robbins Instrument Corporation)

three minutes is in a pregangrenous state and shows lack of collateral blood supply. After a paravertebral block with procaine, a more rapid filling time may take place

When intermittent venous hyperemia is going to be used for treatment as an adjunct of surgical therapy, the venous hyperemia is maintained, not only until the surface veins begin to fill, but until they are maximally distended and until a marked rubor of the digits appears This time, anywhere from one to eight minutes is used for constricting the extremity, whereas during release the rubor should disappear and the veins must empty While the treatment by intermittent venous hyperemia will be discussed in part V, it is mentioned here only in order to emphasize that the venous filling time of an extremity is not identical with the time of compression used in rhythmic constrictors During this treatment a prolonged stretch of the venocapillary bed is sought and more time is needed to develop a high venocapillary pressure throughout the limb

Reactive Hyperemia

The venous filling time cannot be used in cases of arteriovenous aneurysm, since the venous bed can hardly ever be emptied. In such extremities,

however the question of adequate circulation arises after the main artery is obstructed in case excision of the fistula is planned without restoration of continuity It is well to wrap the extremity in a warm wet towel to minimize increased vascular tone or to immerse the lower leg and foot in water at 95 F for 10 minutes The extremity is maximally elevated and an Esmarch or Martin bandage is placed around the root of the limb with such pressure as to obliterate all peripheral pulses distal to it If available a large blood pres sure cuff can be used preferably inflated with great rapidity to produce an ischemic field in the extremity (fig. 30) Suprasystolic compression is main tained for five minutes after which the pressure is suddenly released A bright red flush will spread distally from the cuff within five seconds and should reach the digits in no more than 10 seconds Sometimes a hesitation is seen at one or another digit as if localized vasospasm or organic obstruction were delaying the appearance of the hyperemia If one is interested in the extent of collateral circulation the main artery should be compressed at the site of the aneurysm before the cuff is released If hyperemia now is unsatisfactory additional methods must be used to increase collateral circulation or arterial repair must be seriously considered This test should never be used in patients with obliterative vascular disease in whom it causes a lot of pain and may even produce damage The slow inexperienced use of an oscillometer on the thigh brought on an acute muscle infarct in the flexor muscles of the calf in a patient who entered the hospital with an acutely thrombosed popliteal aneurysm

2. GENERAL CARDIOVASCULAR STUDY

Methods to ascertain the patient s vascular status should continue with a search for involvement in other regions of the body An examination of the eyegrounds an electrocardiogram a two-meter chest film and a renal con centration test serve a most useful purpose Occasionally an electroenceph alogram is studied Not only do these examinations help to establish the diagnosis of the local impairment of circulation but they allow an estimate of the extent of involvement and thus indicate or prohibit certain surgical procedures It is not within the scope of this monograph to discuss the value of these methods in detail only a few general statements can be made based on personal experience Obviously one has to rely heavily here on the co-operation of other specialties

The Eyegrounds

A well trained ophthalmologist examines every hypertensive patient who is a candidate for sympathectomy The grading used is that of Keith and Wagener and its modification by Sanford R. Gifford whose untimely death deprived us of his vast experience and ingenuity Arteriovenous nicking and slight tortuosity of the arterioles, silver wire sclerotic arterioles hemorrhages

and exudates and finally papilledema are looked for The correlation of these findings with the involvement of other vessels will be discussed in chapter 15, Hypertension Diabetic retinopathy has certain characteristics which have been fully discussed by Wagener[15] and Ashton [16] In addition to sclerotic lesions, there seems to be evidence here of increased capillary permeability, of venular thromboses, of small venous aneurysms running a parallel course with peripheral neuropathy of the diabetics, and capillary glomerulosclerosis of the kidney (Kimmelstiel-Wilson's syndrome) Lipemia, with and without diabetes, is readily discernible by its color in the retina, if it is high enough

Congenital vascular anomalies, especially if they present themselves on the head and face, always call for fundoscopy, since angiomatous malformations may be seen in the eyegrounds This is part of the picture of Lindau-von Hippel disease Arteriovenous communications between the carotid artery and internal jugular vein may show venous pulsation in the retina A venous pulse either means an arteriovenous communication or a venous hypertension, since the capillary pulse or the pulse of an adjacent artery is transmitted to the distended vein

Buerger's disease (thromboangiitis obliterans) seldom shows vascular changes in the retina, although Marchesani[17] has made a special point of the white perivenous sheathing which ophthalmologists associated with our group have never found to be diagnostic However, evidence of central angiospastic retinopathy as described by Gifford[18] is not uncommon, especially in heavy smokers

In peripheral vascular sclerosis, sclerosis of the retinal vessels is very frequent When this is advanced, cerebral arteriosclerosis is to be expected and may influence one to advocate conservative surgical procedures, such as sympathectomies for peripheral vascular disease instead of replacement of closed vessels by grafts Unfortunately, however, retinal and cerebrovascular involvement often do not run a parallel course The reactivity of retinal vessels to 100 per cent oxygen seems a promising method for detecting early vascular sclerosis in the retina [19]

In retinitis pigmentosa, a hereditary neurodegenerative disease, there is considerable narrowing of the retinal vessels Sympathetic block to the homolateral stellate ganglion may increase the size of the vessels This may be difficult to measure even with a Morgan grid However, a striking temporary improvement of visual activity has been observed by Gamble following such a block in one of our own cases * Such a finding might suggest a beneficial effect of sympathectomy on retinitis pigmentosa seen early, although Gifford and the author could not find any improvement in the late cases [20]

Generally speaking, an examination of the vascular status should include the study of eyegrounds, a detailed study then is made by the ophthalmologist in case of abnormal findings

* Unpublished observation

The Electrocardiogram

Again no attempt is made here to discuss certain patterns which are significant in vascular disease This is the field of the cardiologist or internist interested in cardiovascular disease Purely from the standpoint of surgical treatment, a few general statements can be made which help to influence the surgical management of certain cases

Electrocardiograms are taken routinely on all patients undergoing surgical treatment for vascular disease Naturally a more thorough physical examination is obligatory preceded by questions concerning congenital lesions rheumatic heart disease attacks of angina attacks of coronary insufficiency and symptoms of heart failure or diminished cardiac reserve In the absence of exertional angina or dyspnea a Master test consisting of an electrocardiogram after graded exercise may reveal abnormal patterns

In arteriovenous aneurysms evidence of heart strain is frequent In thromboangiitis obliterans, 50 per cent of our patients showed an abnormal electrocardiographic pattern [21] In peripheral vascular sclerosis myocardial changes are frequent None of these findings contraindicate an operation Only when serial tracings show a recent myocardial infarct is operation to be postponed for 9 to 12 weeks or entirely to be abandoned Pulmonary embolism, while it is supposed to show a very distinct pattern can not be often differentiated from that of acute coronary occlusion [22] Progressively increasing myocardial damage when previous tracings are available from one s own files or from those of others would also limit surgical activity but of course other factors have to be weighed No cardiographic pattern can ever make the diagnosis of clinical heart disease and the opinion of an experienced internist is routinely sought in such situations Certainly with a badly damaged myocardium and a short life expectancy extensive surgical procedures such as aortic resections or even a lumbar sympathectomy are ill advised Here a chemical sympathectomy with phenol may have to be substituted for the surgical removal of the chain

Chest Films

The size of the heart and thoracic vessels as expressed in these films is of much aid In congenital cardiovascular anomalies the configuration of these structures and their fluoroscopic appearance should be studied The surgeon can learn a great deal by attending the fluoroscopic examination instead of relying on a typewritten report In traumatic arteriovenous aneurysms, the heart enlarges, depending on the size and duration of the fistula The surprising decrease in the size of the heart after closure of a fistula has been often stressed (fig. 31) So does the hypertensive heart decrease after successful sympathectomy (fig 32) In a myocardial occlusion of some magnitude, fluoroscopy shows a rigidity of the infarcted segment or a paradox ballooning when the rest of the heart muscle contracts in systole A kymographic tracing is more conclusive however In luetic aortitis and in luetic

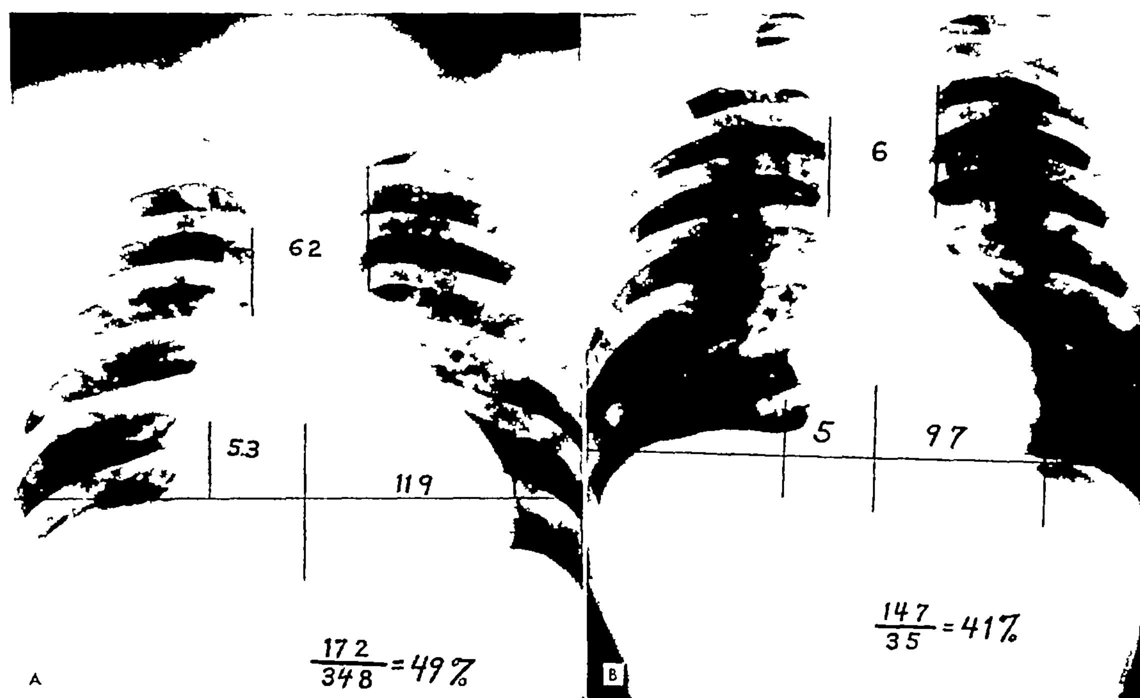


FIG 31 Two meter chest films before (A) and after (B) repair of multiple arteriovenous fistulae following a gunshot wound The cardiothoracic ratio has notably decreased

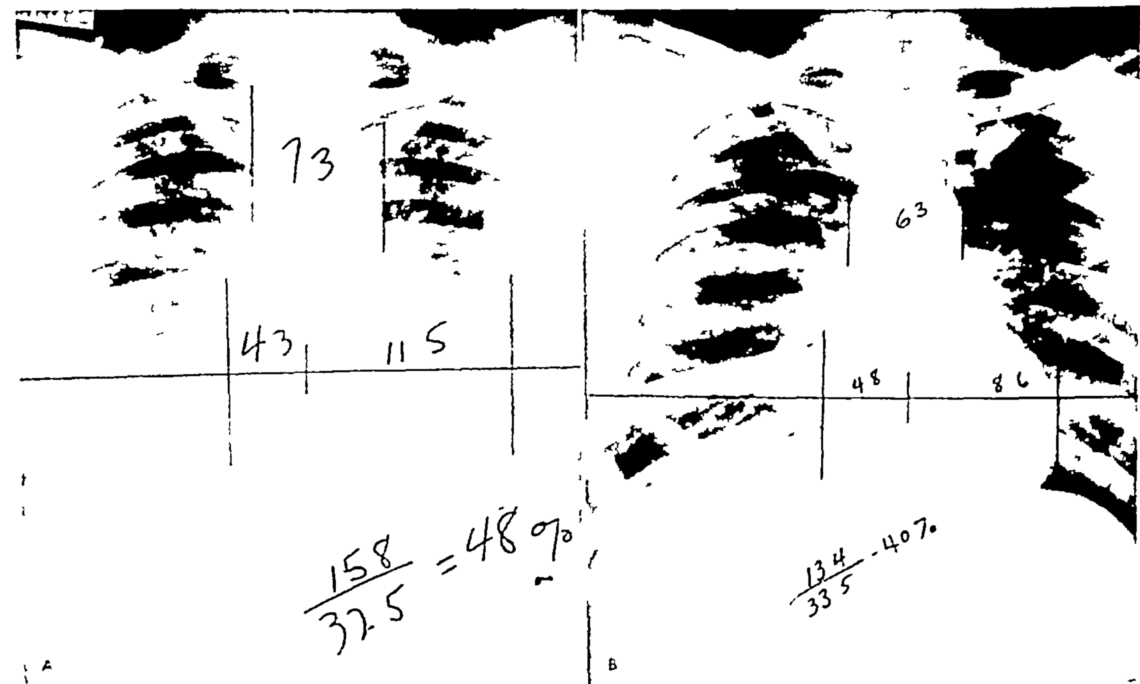


FIG 32 A and B Two meter chest films in a hypertensive diabetic on digitalis Following bilateral splanchnicectomy, digitalis could be discontinued The cardiothoracic ratio dropped from 48 per cent to 40 per cent There was a five year follow-up



FIG. 33 A thoracic aortogram in L.S., a patient with a luetic aneurysm of the descending aorta This was successfully resected by Dr W S Dye

and arteriosclerotic aneurysms of the aorta anteroposterior lateral and oblique films reinforced by a barium swallow supply much information regarding the advisability of surgical treatment. The differential diagnosis between an aneurysm of the aorta and a mediastinal tumor is difficult even after the lesion is palpated through the open thorax Angiocardiogram or a thoracic aortogram may aid in the diagnosis (fig. 33)

Concentration Test of the Urine

For practical purposes and in the nonhospitalized patient, the study of the specific gravity of the urine after withdrawal of water from 6 p m to 8 a m is very helpful, not only to rule out serious renal impairment, but for purposes of follow up studies after dorsolumbar sympathectomies for hypertension Cardiovascular renal disease may affect renal circulation more intensively in some patients and in undertaking any treatment for a diffuse lesion as essential hypertension, it is well to have an idea of the vascular status of the kidneys The concentration test (and no dilution test at this time) simply serves as an orientation The method of sizing up renal function in hypertensives will be discussed in more detail in chapter 19 In peripheral

vascular sclerosis it is well to be aware of the reserve functional capacity of the kidney as measured by this test Aortic thromboses or aneurysms may encroach on the lumen of the renal arteries A 15 minute P S P excretion may give additional information Less than 15 per cent excretion in 15 minutes indicates extreme caution in manipulating the renal vessels

Blood Chemistry

It is of great value to estimate the nonprotein nitrogen, urea nitrogen, the blood cholesterol and the blood sugar of all patients whose cardiovascular system is under study Naturally, when other questions arise, such as those of gout, protein depletion and vitamin deficiency, other tests become necessary However, the four previously mentioned tests are *always* determined, since they give frequent and often unexpected information

The ratio of *urea nitrogen* to *nonprotein nitrogen* $\left(\frac{\text{urea N x 100}}{\text{N P N}}\right)$ is a valuable index of renal function, often when toxic, septic patients are admitted to the hospital requiring amputation, this extrarenal azotemia is high It is surprising how these values may return to a normal or slightly above normal level when absorption of this toxic, sloughing material is eliminated or the patients are rehydrated.

Blood cholesterol levels above 250 mg per 100 cc of blood are not infrequently encountered They occur in diabetes, but also in patients with hypertension or peripheral vascular sclerosis In a questionable case this finding would suggest atheromatosis The rise in blood cholesterol may, of course, be due to a large number of factors, but it may well be that its steady high level predisposes to atheromatous deposits Cholesterol-phospholipid ratios or the fat tolerance developed in the pathological laboratory at St Luke's

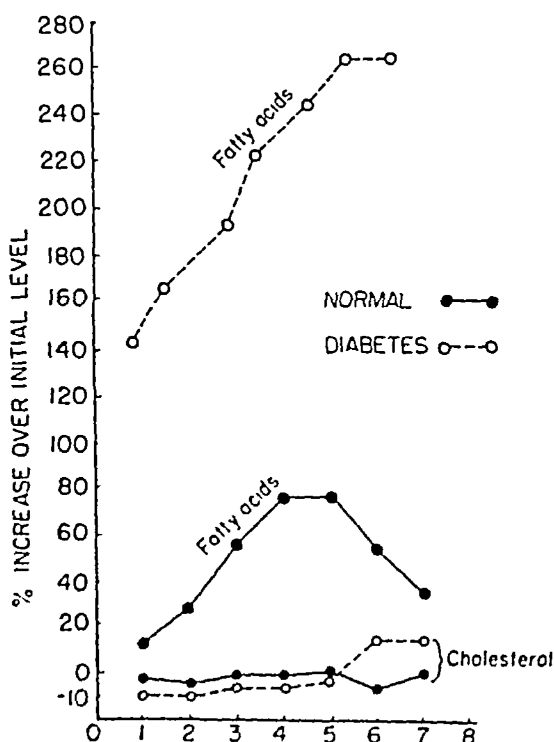


FIG 34 Fat tolerance curves in a normal and a diabetic individual Note the rise and delayed return of fatty acids in the diabetic after ingestion of a fat meal, the cholesterol does not rise (Hirsch, E F and Carbonaro, L Serum Esterified Fatty Acids with Fat Tolerance Tests in Diabetes Mellitus A M A Arch Int Med , *86* 519, 1950)

Hospital offer great promise [3] (fig 34) For this reason an effort should be made to control the lipid metabolism postoperatively by a low fat diet by thyroid and by iodine Lipotropic drugs have not been used on our service

Blood sugar may be higher than normal in patients suffering from cardiovascular renal disease without fasting glycosuria when the renal threshold for sugar is high or it may be within normal limits and yet a glucose tolerance curve shows a diminished tolerance to sugar a diabetic type of curve Sugar tolerance curves are not determined on every patient suffering from hypertension or peripheral vascular sclerosis but a surprising number of patients in this group above 50 years of age have shown abnormal curves A single postprandial blood sugar taken two hours after breakfast or lunch is an excellent screening procedure and preferable to a fasting one

3 TESTS FOR VASCULAR TONE

Reflex Dilatation To Heat (The Landis-Gibbon Test)[24]

When one extremity is submerged in warm water preferably between 110 and 115 F and kept there for 30 minutes the contralateral extremity in fact all three other extremities show vasodilation Immersion of the two upper or two lower extremities gives an even better reflex vasodilation This is a simple method to test the capacity of a regional vascular bed to dilate, although severe vasospasm is sometimes not released The mechanism of this phenomenon seems to depend on a warming of the blood which drains the submerged extremity but a cutaneous reflex is also involved When the blood reaches the vasomotor center reflex sympathetic inhibition together with active sympathetic vasodilation occurs Indirect vasodilation is present when the heated area is sympathectomized or even totally denervated but the rise in temperature or increase in blood flow in the opposite extremity does not take place when it is deprived of its sympathetic nerve supply When the return flow of blood from the heated extremity is slow because of impaired arterial or venous circulation incomplete sympathetic inhibition takes place For this reason the routine use of this test has now been abandoned, but the reflex is of great help in the treatment of vascular disease since either a large heat cradle over the abdomen or lumbar diathermy[25] will produce as much increase in blood flow of the lower extremities as if they were heated directly [26] This latter procedure however is to be condemned in any vascular disease where arterial circulation is diminished The Landis-Gibbon test can be used to test for adequate sympathetic denervation [27] To simplify the test one can use a finger plethysmograph to indicate whether or not the effect of reflex heating has been inhibited by a complete sympathectomy

Peripheral Nerve Block

When a mixed peripheral nerve carrying vasomotor efferent fibers is blocked with procaine a vasomotor paralysis together with anhidrosis will

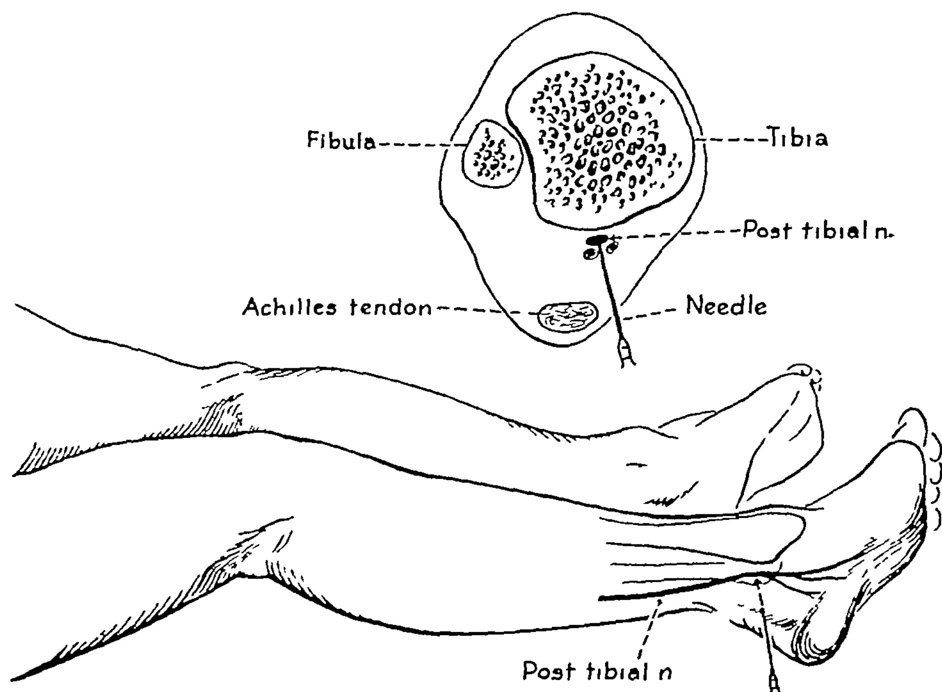


FIG 35 Block of the posterior tibial nerve at the inner malleolus The patient lies on his side, with the extremity to be injected on the lower side, the Achilles tendon faces the injector An intradermal wheal is raised at the inner surface of the tendon and a 3-inch 22-gauge needle is slowly inserted in a postero-anterior direction, injecting 1 per cent procaine solution in advance of the needle Paresthesia, radiating toward the heel and the sole of the foot, is signalled when the needle arrives at the perineurium of the posterior tibial nerve 5 cc of 1 per cent procaine are injected at this site Within 20 minutes, the heel, the sole of the foot and the plantar surface of the big toe should be anesthetic Testing for increase in temperature may now begin

develop within 10 to 20 minutes The area corresponds to the cutaneous sensory distribution of the nerve In the lower extremity the posterior tibial nerve can be readily blocked midway between the internal malleolus and the Achilles tendon (fig 35), whereas on the upper extremity the ulnar nerve at the elbow (fig 36) or the median nerve at the wrist can be injected In a room of moderate temperature (68° F to 74° F) the skin temperature of the area to be desensitized is taken several times before and every 10 minutes after the injection If the skin is not anesthetic, the block has not been accomplished Full vasodilatation should be accompanied by a skin temperature of at least 31 5° C Failure of the temperature to rise indicates severe terminal arteriolar obstruction, giving a poor prognosis In many types of peripheral vascular disease there is a combination of organic obstruction with vasospasm superimposed on normal vasoconstrictor tonus, and in these a partial rise in temperature occurs Morton and Scott[28] have called the difference between the full and partial level of vasodilatation the vasodilator index, and this is a useful yardstick of the capacity of the vascular bed to dilate

This method is simple, readily carried out on ambulatory patients and can be easily combined with oscillometry or finger plethysmography However, certain drawbacks are obvious Since it is performed at a peripheral level, peripheral nerve block gives no information regarding higher cross sections of the extremity Thus, suppose one is dealing with an embolus to

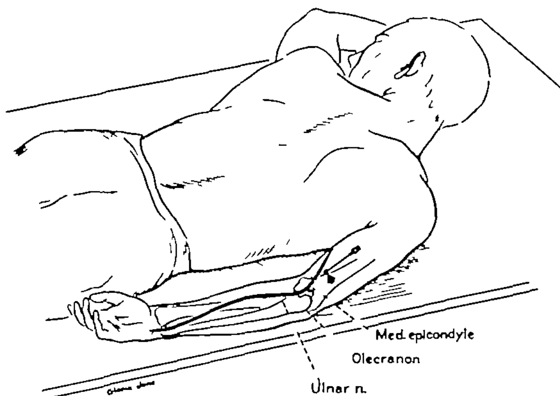


FIG 36 Ulnar nerve block at the elbow The patient is in a prone position, with his arm resting alongside his body With the thumb and index finger of the left hand, the ulnar nerve can be grasped above the ulnar groove between the olecranon and the internal condyle of the humerus. The intradermal wheal is raised about an inch and a half above the bony landmarks. A 2 inch 25-gauge needle is inserted parallel with the nerve and the shaft of the humerus. Paresthesia must occur along the ulnar distribution toward the fourth and fifth fingers. 5 cc. of 2 per cent procaine are now injected. Tests for vasodilatation are to be carried out in 20 to 30 minutes, but only if the fifth finger and the ulnar side of the fourth finger have become anesthetic.

the femoral artery which has led to severe ischemia of the big toe, or to a segmental stenosis of the iliac artery The element of vasospasm in this case may affect the entire iliofemoral segment, possibly the entire arterial and venous bed, which will not be influenced by blocking the posterior tibial nerve at the ankle Another objection to the method is that since it blocks not only sympathetic but also sensory fibers it gives no information about the relief of pain to be expected when sympathectomy is performed It is then not an ideal preoperative test for sympathectomy

The use of this test has been limited to patients in whom paravertebral sympathetic block is anatomically impossible as in orthopedic lesions of the spine or in whom sympathectomy has already been performed In the latter case should a rise in skin temperature occur it would indicate an incomplete operation or regeneration of sympathetic fibers

Paravertebral Sympathetic Block

This test is the most direct approach to the problem as to whether an increased or even a normal vasomotor tonus contributes to the circulatory embarrassment of the area under study With certain precautions the method

is safe, painless and harmless It is true, however, that a careless technique has led to some difficulties and repetition of the test after the patient has been hurt or frightened once before may be difficult or impossible It is obvious that it can never reproduce the full effect of sympathectomy since it may take six months to a year to obtain the maximum effect of this operation, and a temporary sympathetic paralysis is not accompanied by the return of intrinsic tone of the vessels (chapter 3)

The patient should be sufficiently prepared for the test with barbiturates One hour before the block 1½ to 3 gr of Nembutal are given and skin temperatures are read on the digits or any special region under scrutiny First, intradermal wheals must be made with the finest hypodermic needles The paravertebral needles for the dorsocervical block must be 4 inches long with 22 gauge, and those for the lumbar sympathetics are 6 inches long with a 22 to 20 gauge needle, preferably the former. A baby spinal needle can be used for dorsocervical block, but the lumbar block should never be attempted without special needles (part IV)

While true procaine sensitivity is uncommon, every patient should be told about receiving a procaine injection, and should he or she state that previously a reaction took place, an intradermal wheal with 1.100,000 procaine should be first produced Most of the so-called procaine reactions occur during dental work or tonsillectomies and are due to too much epinephrine in the solution Nevertheless, true procaine sensitivity does exist and may produce severe sensitization reactions, if not death Among many thousand paravertebral blocks done by our group, two near fatal reactions have been seen Such patients then should be tested with Metycaine hydrochloride or Xylocaine hydrochloride, if the test is of great importance

The Anterolateral Stellate Block

For releasing the vasoconstrictor tonus of the head and neck, the stellate block of Leriche[29] performed from the front is preferable to the dorsal approach (fig 37) We also use this approach in emergencies, as in traumatic or embolic vascular occlusions of the upper extremity or brain, when the patient is severely ill, flat on his back or unconscious

With a small pillow under the patient's neck and his head rotated toward the opposite side, the transverse processes of the lower cervical spine are palpated and a line is drawn from the mastoid process through them as far as the clavicle 2 fingerbreadths above the clavicle a dermal wheal is made and a 4-inch 22-gauge needle on a 5 cc syringe is inserted One per cent procaine is slowly but continuously injected ahead of the path of the needle, so as to minimize discomfort The needle must touch the tip of the sixth transverse process, and this is the first landmark After this, the needle must gently slide along or above the transverse process until it makes contact with the body of the vertebra Aspiration must be made for blood, spinal fluid or air from the pleural cavity, and if this reveals no such difficulty 10 cc of 1 per cent procaine without epinephrine is injected The proof of a satisfactory

injection can be recognized by the appearance of a Horner s syndrome (miosis ptosis, enophthalmos) cessation of sweating over one side of the face neck and upper extremity and an increase in surface temperature over the same area Some individuals develop a marked ptosis of the upper lid and a great deal of conjunctival hyperemia Others note the dryness of the nasal mucosa on the injected side or a great deal of warmth in the hand or fingers

If the needle is not posterior to the great vessels of the neck, a huge hematoma may develop if the needle is not directed in a posterior and cephalad direction, the pleura or even the lung can be punctured A small pneumothorax is quickly absorbed and may be asymptomatic but in the course of the last 15 years we have seen residents puncture the lung One massive collapse of the lung did not expand for eight weeks leaving residual fluid in the costophrenic angle another patient had hemoptysis for several days after an anterior stellate block I have also seen a lateral spinal puncture and a fatal hemorrhage into the cord in one patient and a high spinal anesthesia with instant death in another patient Both of these injections were done by careful specially trained assistants Such untoward reactions must be minimized especially if the block is not a therapeutic but a diagnostic one On a vascular surgical service several members of the team must have mastered

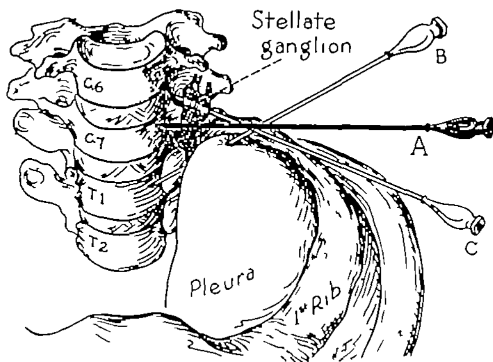


FIG 37 Anterolateral block of the cervical sympathetic chain. (A) The cervical position of the needle, locating the lateral surface of the seventh cervical vertebra or the disc between the sixth and seventh vertebrae. Note that the direction of the needle is at right angles to the spinal column. (B) This needle is directed caudally often for the search of the first rib which is unnecessary Pleural puncture or lung puncture may follow (C) This needle, if directed cephalad, may pierce the dural covering of the roots or enter the intervertebral foramen and puncture the cord. (Gilbert and de Takats Emergency Treatment of Apoplexy J.A.M.A., *136* 659 1948)

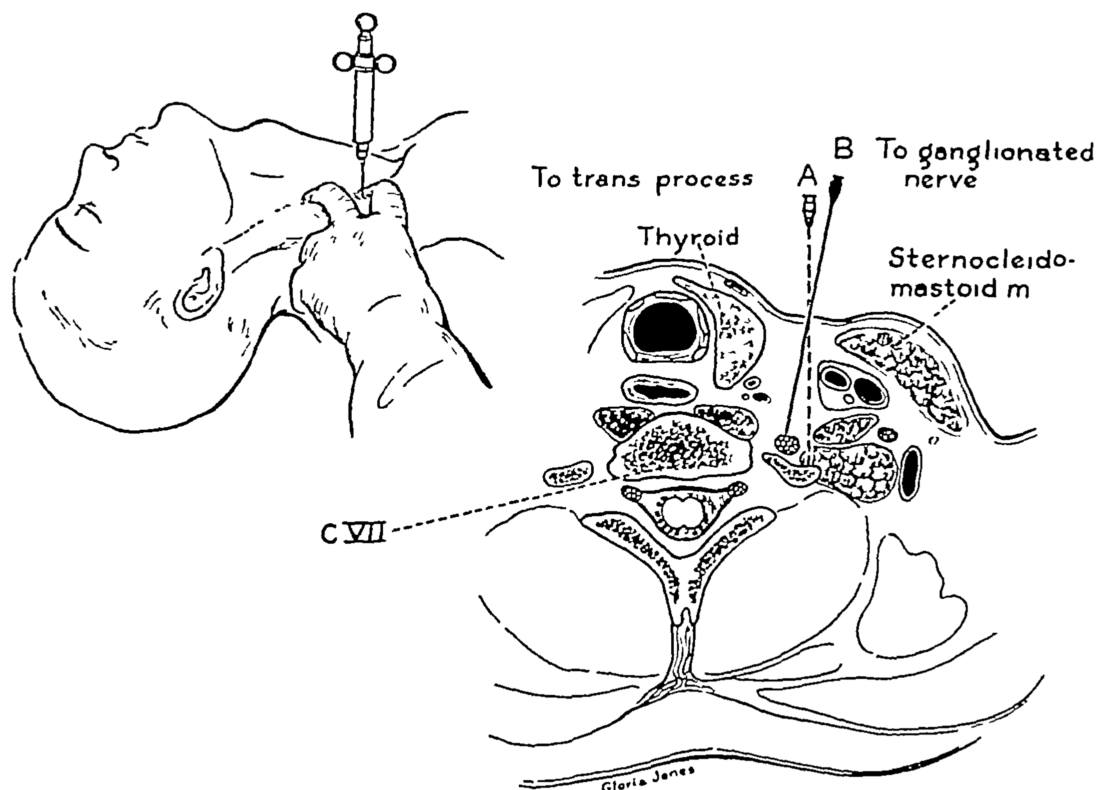


FIG 38 An intradermal wheal is placed 2 fingerbreadths lateral to the jugular notch and 2 fingerbreadths above the clavicle The sternocleidomastoid muscle is retracted laterally and downward, and a 2-inch 22-gauge needle is inserted in an anteroposterior direction so as to gently touch the seventh transverse process After the bone is reached, the needle is slightly withdrawn and reinserted more medially 5 cc of 1 per cent procaine solution are slowly deposited

the technique, but all rotating surgical residents or interns should not be permitted to perform these blocks except under supervision This is true of all sympathetic blocks, but especially of the cervicothoracic type

A detailed description of indications and technique of the stellate block is given in a recent monograph by D C. Moore [30] He, as most anesthesiologists, prefers the anterior block to the ganglionated chain, and many of our residents have preferred this shorter route which has almost entirely replaced the previous method, at least in our hands (fig 38)

With the second and third fingers of the left hand, downward and lateral pressure is exerted on the sternocleidomastoid muscle and the carotid sheath The pulsation of the carotid artery must be well felt by the tips of the fingers. With the right hand an intradermal wheal is raised, 2 fingerbreadths lateral to the jugular notch and 2 fingerbreadths above the clavicle This landmark should lie over the seventh transverse process

The patient lies in the dorsal recumbent position with no pillow under his head or neck With a 10 cc syringe attached to a Luer-Lok, 2-inch 22-gauge needle, procaine is gently injected in an anteroposterior direction At the depth of 1½ to 2 inches, the transverse process is reached The needle now is slightly withdrawn and aspiration is made for blood or air. If none appear, 1 cc of 1 per cent procaine is injected If dizziness appears immediately the injection has been intravascular, perhaps even in the vertebral

artery If no reaction occurs a total of 5 cc of 1 per cent procaine are deposited Horner s syndrome should appear in 10 to 15 minutes if not sooner

The cervical sympathetic block has been used extensively on our service for the emergency treatment of certain types of apoplexy It is regarded as a test of available collateral circulation to the infarcted area and if followed by immediate improvement is then used for therapeutic purposes daily alternating on both sides [31]

The Posterior Approach to the Dorsal Sympathetic Chain

For certain diagnostic procedures for testing the circulation of the upper extremity and for desensitization of the heart or aorta in coronary sclerosis or aortic aneurysm this is the method of choice (fig 39) The patient is in a

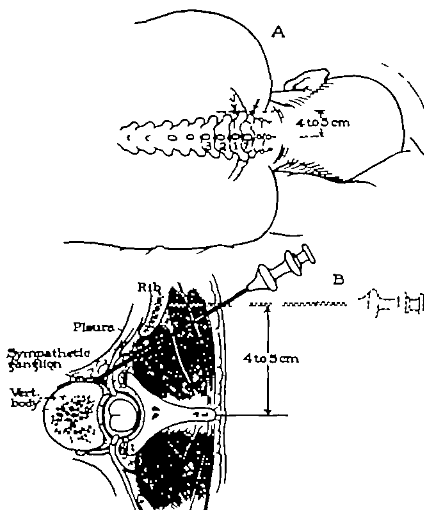


FIG 39 Paravertebral block of the dorsal sympathetic chain. Note the positions of the needle. In order to get farther away from the intercostal nerve and the intervertebral foramen and closer to the ganglionated trunk, the tip of the needle can be so rotated that its beveled tip is turned away from the bone and can be advanced for another centimeter On injecting 5 to 10 cc. of 1 per cent procaine, vasodilation in the hand should be rapid. Horner's syndrome develops if the stellate ganglion is blocked but injections to the upper dorsal ganglia will not affect the oculopupillary outflow (Military Surgical Manual, Vol. V W B Saunders Co., Philadelphia, 1943)

lateral position, the head well flexed over the thorax The prominent seventh spinous process is marked with tincture of iodine or 2 per cent brilliant green, then the next three or four dorsal spinous processes are identified and marked From these processes, lines are drawn at right angles to the axis of the spine and 3 fingerwidths lateral to the spinous processes another mark is made opposite to the lower level of the second, third and fourth spinous process Intradermal wheals are made here with procaine and the 4-inch 22-gauge needle is gently inserted at right angles to the skin until it reaches the rib or the costovertebral junction Then the needle is slightly withdrawn and with an angle of 30 degrees toward the midline and slightly cephalad, it is reinserted so as to reach the body of the vertebra, it is pulled back again and insinuated slightly forward so as to get to the lateral border of the vertebra, since the first point reached is usually too far posterior to the chain Aspiration is now made for blood, spinal fluid or air, and in case this is negative, 5 cc. of 1 per cent procaine are injected at each level This should produce a complete temporary sympathetic denervation of the upper extremity When the purpose of the injection is to block the sensory supply to the heart, the fourth and fifth segments should also be injected It is best not to inject both sides at once but on different occasions, absorption of procaine is rapid from the paravertebral spaces and procaine may get into the spinal canal if the faulty angle of the needle brings the fluid to the intervertebral foramen Procaine may also extend in the perineural sheath toward the spinal canal

Such paravertebral injections may be done at any segment of the dorsal spine depending on the area under examination or treatment To relieve pain in an aneurysm of the descending aorta, it may be necessary to inject the lower six segments For diagnosing the visceral origin of pain or to break up a visceral sensory-sympathetic reflex, Table II gives the segments to be

Table II

DIAGNOSTIC PARAVERTEBRAL BLOCKS FOR VISCERAL PAIN OF THE ABDOMEN*

VISCUS	SEGMENTS INJECTED
Liver and gallbladder	D-7—D-9, right
Stomach	D-6—D-8
Duodenum	D-7, right
Kidney	D-12—L-2, unilateral
Appendix	D-12—L-2, right
Pancreas	D-8—D-10, unilateral or bilateral

* Combined experience of Läwen, Mandl, Kappis, J C White and de Takats

injected From a vascular standpoint, the release of reflex anuria by blocking D-12, L-1 and L-2 segments should be stressed, since this effect seems to depend on the release of a massive vasoconstriction of the afferent arterioles to the glomeruli

Other effects on esophageal, gastric and biliary colics seem to be due to release of sphincteric action or to the blocking of viscero-sensory fibers, and

need not be discussed here White Smithwick and Simeone have dealt with this subject in detail [3]

The Lumbar Sympathetic Block

This is by far the most frequent and technically the easiest block (fig. 40) and yet a certain percentage of failures is to be expected because of occa

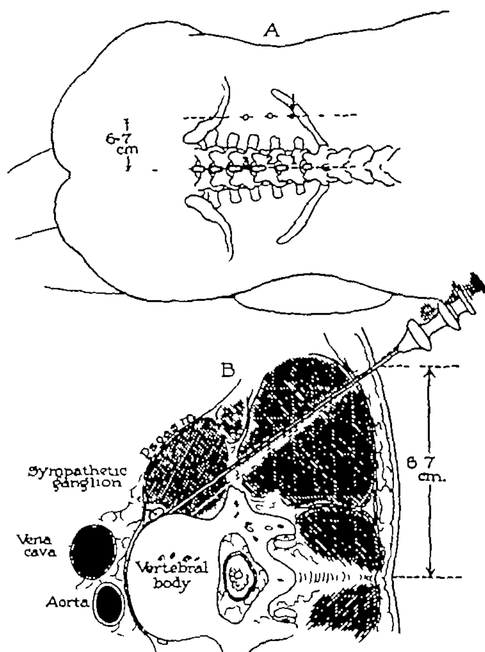


FIG 40 Paravertebral block of the lumbar sympathetic ganglia. This approach is through one wheal, placed at the edge of the erector spinae muscles, below the twelfth rib It is practically the same approach used for aortography except that puncture of the aorta or vena cava are scrupulously avoided. While the lateral position of the needle is more apt to cause a spinal puncture, this is the only way to get anterior to the somatic nerve supply and close to the ganglia (see the text for a description of the procedure) (Military Surgical Manual Vol. V., W B Saunders Co., Philadelphia, 1943)

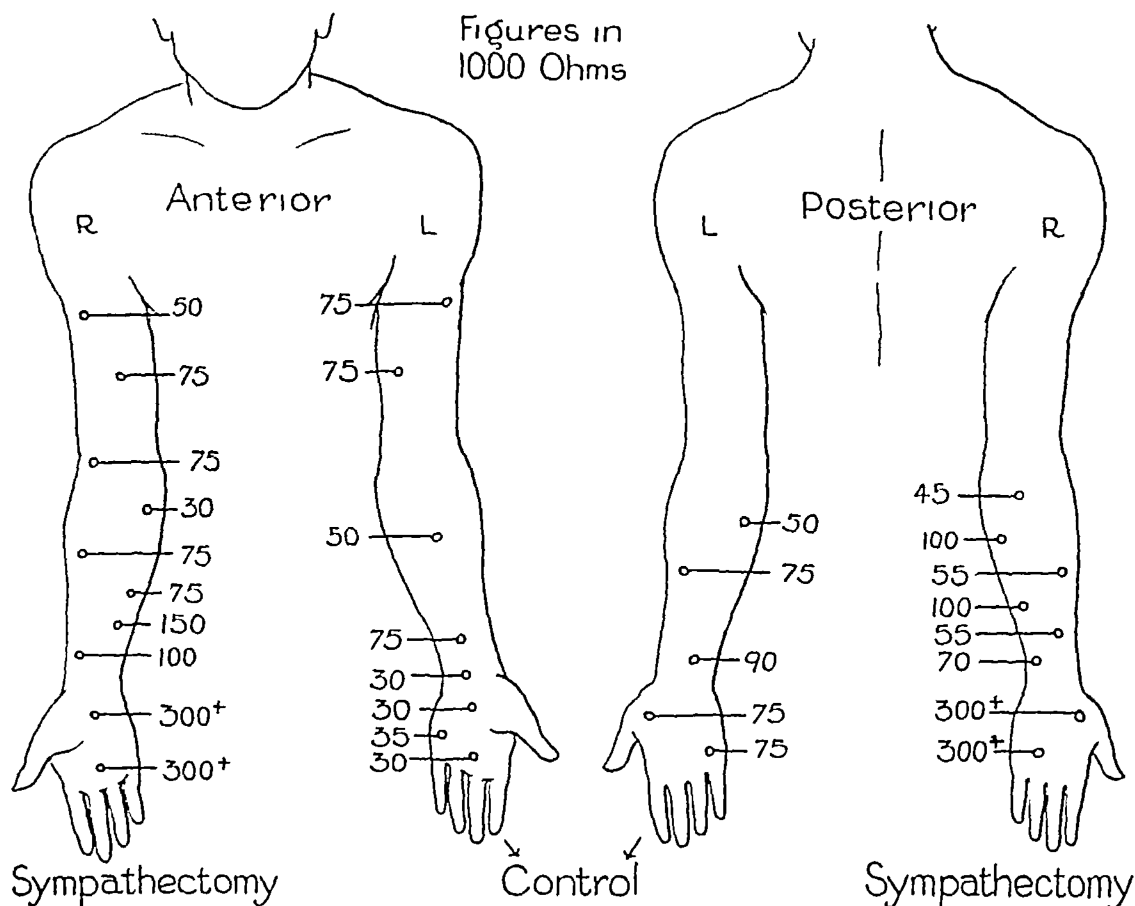


FIG 41 Palmar sweating has been abolished in the right hand, skin resistance rose to more than 300,000 ohms in the right palmar and dorsal surfaces The record indicates that this sympathectomy is incomplete (done in 1936 from an anterior approach), the third and fourth dorsal ganglia have not been removed Recurring sweating occurred two years later and was abolished by a dorsal sympathectomy.

sional anatomic difficulties The patient should be on his side with a pillow under the lumbar region, his back flexed and the thighs drawn up on the abdomen just as for a spinal puncture A line is drawn with tincture of iodine or 2 per cent brilliant green from one anterior superior spine to the other, where this line bisects the spinal column, the spinous process or the interspace between the fourth and third spinous processes will be found The four lumbar spinous processes are marked and horizontal lines are drawn laterally until, at a distance of 4 fingerwidths from the midline, the intradermal wheal is made Four injections are made now, one for each segment, using the same method as described for the dorsal sympathetic chain Here the 6-inch 22-gauge needle first has to reach the transverse process, afterward it is turned inward and cephalad to touch the body of the vertebra, finally a third contact is sought farther anterior than the second one 10 cc of 1 per cent procaine solution are injected into each of the four segments For practical purposes, we have not found it necessary to control the position of the needle with roentgen rays However, the beginner may check his first attempts with profit Paravertebral blocks may be done for therapeutic purposes either by repeated injections or by using a long-lasting anesthetic, notably phenol.

Technique and indications for such a procedure will be discussed on page 69

Such an injection if satisfactory should produce complete anhidrosis of the entire extremity The extent of this as measured by electrical skin resistance is shown in figure 41 Often the first lumbar segment remains unblocked but instead the procaine has infiltrated the first lumbar sensory outflow or trickled down on the anterior surface of the psoas muscle (fig 42 A and B) In such a case numbness of the anterior surface of the thigh and weakness of the psoas or quadriceps muscles may result These are all temporary and will promptly disappear with the absorption of procaine Occasionally through a lateral spinal puncture spinal anesthesia results which naturally alarms everyone but which subsides within a few hours This may require hospitalization of the ambulatory patient

Of course the rise in skin temperature at different levels of the lower extremity is what one is testing and this should be undertaken 30 minutes after the completion of the injection There may be a rise to full vasodilatation a partial rise no rise or even a fall in skin temperature (fig 43) This paradoxical fall in skin temperature after block of the regional sympathetics is a significant phenomenon It was described from our clinic in 1945 in a paper on Buerger s disease [17] and indicates a complete failure of the terminal vascular bed of the digit to relax and a diversion of the small amount of blood present to areas where peripheral resistance is lower It indicates a pregangrenous state and should sympathectomy be undertaken, this area

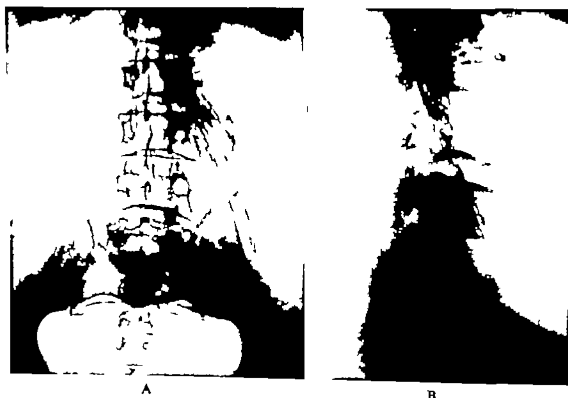


FIG 42. (A) An anteroposterior view of ½ cc. of Lipiodol deposited paravertebrally to the second lumbar segment and trickling down the psoas muscle (B) lateral view of the same injection

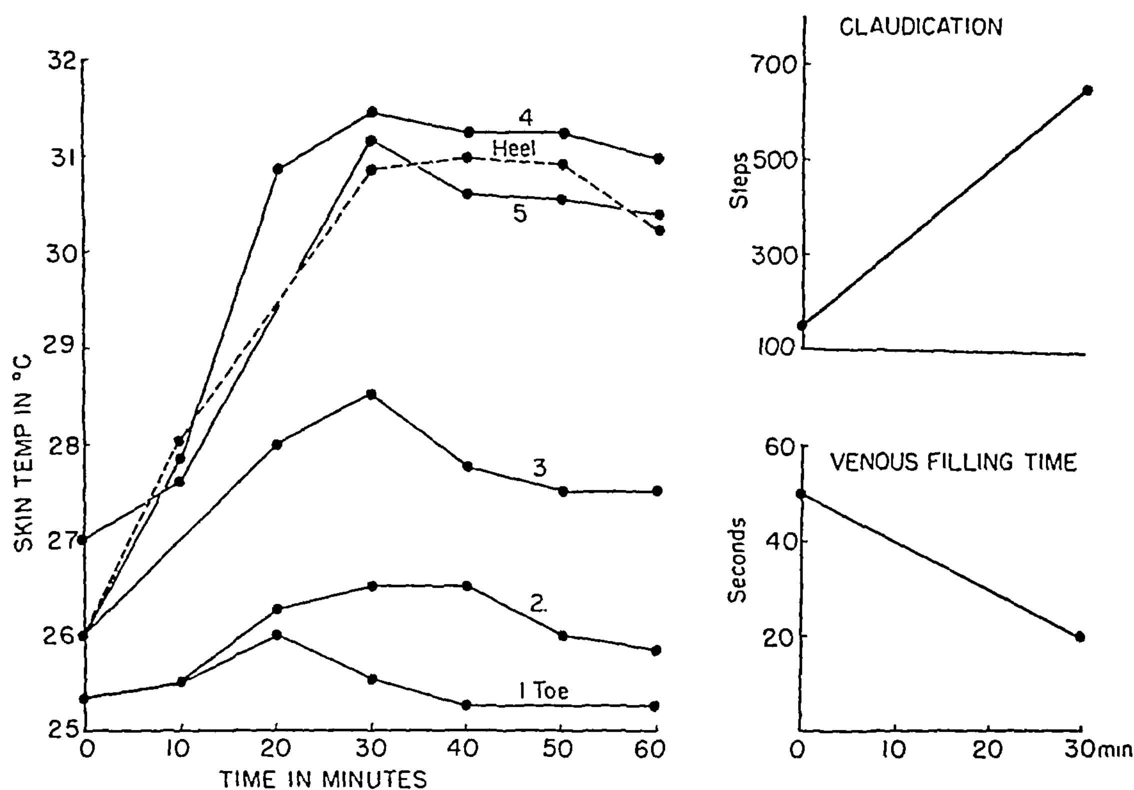


FIG 43 The effect of lumbar sympathetic block on skin temperatures, claudication and venous filling time Note a rise to the desired vasodilatation level of 31° C in the heel and fourth and fifth toes The temperature of the third toe rises moderately, that of the second and first toes hardly or not at all These two toes were amputated at the time of sympathectomy The patient's ability to walk without cramping increased from 150 steps to 650 steps The venous filling time shortened from 50 seconds to 20 seconds (de Takats Value of Sympathectomy in Treatment of Buerger's Disease Surg Gynec and Obst, *79* 359, 1944)

may rapidly become gangrenous It is wise for this reason to combine sympathectomy in such cases with a simultaneous amputation of toes having no rise in skin temperature

When lumbar sympathetic block is done not for diagnostic but for therapeutic purposes, as in acute venous or arterial occlusions, in traumatic edemas or for a chemical sympathectomy, the patient tolerates a single injection much better This should be done at the level of the second lumbar vertebra and if the needle is properly placed, 30 cc of 1 per cent procaine will diffuse along the loose paravertebral space to produce a satisfactory vasomotor palsy It should be remembered that in many cases no preganglionic fibers emerge from the cord to the sympathetic ganglia below the second lumbar ganglion, so that such an injection should be completely satisfactory. This method, however, will show more frequent failures, since everything depends on the proper placement of one needle

Other Methods Producing Sympathetic Paralysis

A number of other procedures can be utilized but have been abandoned in favor of paravertebral sympathetic injections Spinal anesthesia produces

not only sympathetic but also motor and sensory paralysis and therefore is unsuitable to imitate the effect of sympathectomy Since it often lowers blood pressure blood flow may actually be reduced in the limb under scrutiny Intravenous triple typhoid vaccine produces marked peripheral vasodilatation it has a central effect like reflex heating of the body and cannot operate in the sympathectomized limb Fever and vasodilatation are preceded by a chill which may be harmful in patients with generalized vascular disease and may lead to thrombosis The test has great historical interest since it was the first one deliberately introduced by Brown and Allen to test for the capacity of the vascular bed to dilate [33] but it has now been definitely superseded by sympathetic block Intravenous Pentothal sodium and inhalation anesthesias produce peripheral vasodilatation and the surgeon may utilize this knowledge if he is operating under such conditions However when induced only for the purpose of testing the peripheral vascular bed these methods seem to constitute an unnecessary hardship for the patient

Intravenous sodium nitrate to test for the flexibility of the vascular bed using an oscillometer was suggested from our clinic,[34] but has also been abandoned With sensitive finger plethysmographs tracings can be taken before and after immersion of the digits into hot water or by using indirect heat or sympathetic block one can demonstrate increased pulsation of the digits If a water plethysmograph is set up many valuable studies of blood flow can be made and many physiologic observations are possible[35] (fig 44)

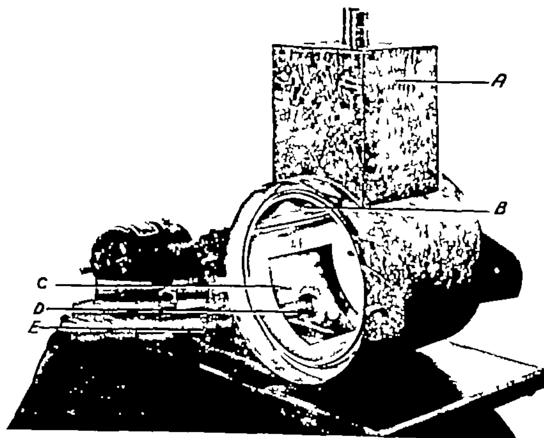


FIG 44 Water plethysmograph modified by D S Miller It was used to study blood flow in causalgic states. (de Takats and Miller Post traumatic Dystrophy of the Extremities. Arch Surg. 46 469 1943)

However, these last two methods are not apt to be available in most hospitals Ganglionic blocking agents, such as tetraethylammonium chloride, hexamethonium, Priscoline and others, produce only partial sympathetic inhibition and have actions other than sympathetic paralysis since they act at ganglionic levels and block parasympathetic impulses There is an amazing difference in response even in young normal individuals [36]

4. ANGIOGRAPHY

There is no part of the vascular tree which has not been explored by angiography Beautiful postmortem angiograms have been presented by Schoenmackers and Vieten[37] but their application to conditions in vivo is limited This entails the injection of a radiopaque substance into an accessible artery or vein and the taking of one or several roentgen pictures, which present a visible pattern of the vascular area under study. Much of the earlier work has been done with Thorotrast, which is painless and does not seem to irritate the intima, but its use is objectionable since the material is retained *in the reticuloendothelial system and gives off gamma rays for many years,* thus producing chronic radioactivity (fig 45) Thorotrast has never been used in our clinics Angiography should not be used unless it supplies information which is otherwise unobtainable. This limits it to certain congenital and traumatic arteriovenous fistulae where a good film may direct the surgical approach, and to some of the chronic, deep venous thromboses in which obliteration of superficial varicosities is debatable The wholesale use of this method for all arterial obstructions and acute or suspected venous *thromboses is not recommended* It usually fails to supply the information where it is most needed It is, however, indispensible before segmental arterial occlusions are resected or reamed out

Angiography can be done by a *single injection*, by *repeated injections*, by *single exposure*, by *repeated serial exposure* and by *cinematography* The last, of course, practiced by Janker in Bonn and K J Franklin in Oxford,[38] permits a truly functional visualization of the living vascular tree However, many years may elapse until it becomes an inexpensive clinical procedure The majority of our films have been obtained by a single injection followed by a single exposure, although one is daily aware of the advantage of two or three serial exposures to better visualize collateral circulation, and most importantly to establish the existence of a patent distal segment If single injections are used, a rough estimate of circulation time can be obtained by the venous filling time

Arteriography

No attempt will be made here to describe the special techniques suitable for each individual vessel For peripheral *arterial visualization*, the brachial, radial and femoral arteries can be readily punctured with an ordinary veni-

puncture needle gauge 16 to 18 attached snugly to a 20 cc syringe A polythene tube with a suitable adapter keeps the hand well out of the field Immediately after bright red waves of blood appear in the syringe the vessel above the site of puncture is rapidly obstructed This can be done by manual pressure in the groin and by a blood pressure cuff inflated above systolic pressure for the upper extremity The solution is now rapidly injected and the first film taken while there is still some material in the syringe Next a few pulse beats are allowed to come through and then suprasystolic pressure is reapplied, a second film is taken and the procedure is repeated a third time The films can be taken in anteroposterior oblique or lateral directions to bring out the desired segment of vessel and to eliminate an overlap of osseous shadows

Often the vessels will not be well visualized especially if the pressure above the artery failed to throttle the blood stream In arteriovenous fistulae the artery empties rapidly into the vein and often the fistula is detectable at the site of greatest venous distention The arterial puncture is painful and its site must be anesthetized with procaine When the solution travels to the arteriolar bed a severe vasospasm with blanching and pain develops For this reason *arteriography should never be done without some type of anesthesia* One can use intravenous Pentothal sodium for the lower and brachial plexus block for the upper extremity Spinal anesthesia for the lower extremities is of too much magnitude and too long duration, since the entire procedure lasts less than a few minutes Anesthesia also has the advantage of relaxing the vascular tree Diodrast produces vasospasm later followed by vasodilation and to differentiate between a spastic artery and an occluded artery on the film is not always easy or possible For a detailed study of the failures errors in interpretation and untoward reactions the studies of Allen and Camp Edwards Lindblom and Passler should be consulted [39]

More and more our tendency has been to expose the vessels to be injected with a small incision under local anesthesia in other words while the percutaneous puncture of the artery can be readily done better pictures are obtained when the vessel is exposed proximally occluded with cord tape during injection and pressure applied on the artery until bleeding from the puncture ceases With percutaneous technique poorer visualization large hematomas and a large abscess requiring multiple aspirations and massive antibiotic therapy have been encountered

Further improvement in femoral arteriograms can be obtained by taking two to three exposures at two-second intervals instead of relying on a single exposure One can expose first the upper half then the lower half of a long 20 by 96 cm. film, use automatic or manually operated devices to obtain serial films or even slide the tube peripherally from thigh to lower leg

Most impressive for clinical use is Pässler s manually operated three cassette arrangement with which he can expose three films at two-second intervals [39b] Lindblom[39e] found that the opaque substance travels 5 to 15 cm a second in the lower extremity without throttling of the blood stream Since the condition of the popliteal bifurcation and the width of the posterior



FIG 45 FIG 46

FIG 45 Thorotrastoma This periarterial injection of Thorotrast was made in another institution eight years before the film was made The patient had a palpable hard mass and considerable femoral and saphenous nerve involvement The roentgenologist mistook the mass for an osteogenic tumor

FIG 46 A normal superficial femoral artery courses through a huge pulsating mass The artery could be saved during removal of the tumor The lesion was a rhabdomyosarcoma

tibial and tibioperoneal segments is so important, we have in a few instances obtained arteriograms by exposing the lower femoral segment in Hunter's canal and studied the vascular pattern of the lower leg which was not, or only insufficiently, visualized by a single exposure following a femoral injection

Because of my belief that all peripheral arterial obstructions, unless a contraindication exists, require a sympathectomy, occlusions of the lower extremities are visualized by femoral arteriograms at the time of sympathectomy under the anesthesia used for sympathectomy Of course, if the femoral pulsation at the groin is weak or absent, an aortogram may need to be considered

The visualization of the superficial femoral artery is easy, that of the popliteal and lower leg arteries is the important problem, and improvement in technique can be expected at this area While a number of arteriograms will be used in later chapters to illustrate clinical entities and their management, a few films may serve here to emphasize technical points

In figure 45 a large opaque mass, the result of extravasated Thorotrast, was discovered by us on a routine flat plate This had been injected eight years before in another institution and still showed radioactivity Organic iodides when accidental extravasation occurs disappear in less than 24 hours from the film The thorotrastoma was painful and produced femoral and saphenous neuritis Figure 46 illustrates a pulsating mass in the thigh which turned out to be a large *rhabdomyosarcoma The femoral artery looks* intact and could be saved at operation Figure 47 is a femoral arteriogram taken on the opposite side to an obviously advanced arteriosclerosis The *artery does not have a smooth contour and shows a few moth-eaten areas* due to plaques It is the picture of early nonstenosing arteriosclerosis There is beginning obstruction visible in figure 48 characteristically at the upper end of Hunter s canal This too is an asymptomatic limb with advanced obstruction on the opposite side Minor procedures such as splitting the roof



FIG 47

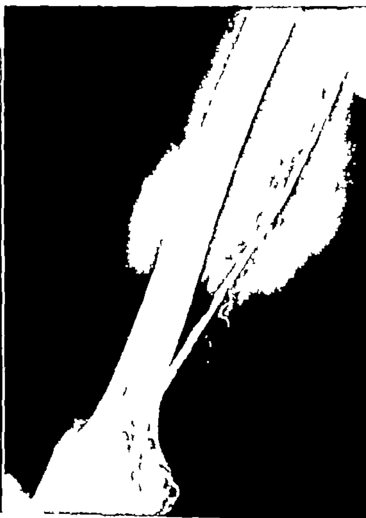


FIG 48

FIG 47 Beginning diffuse atheromatous of the superficial femoral artery *in the case of* F.S a 56 year old man, whose superficial femoral artery was completely obstructed on the opposite side. Note the ragged contour of *the vessel, especially* in Hunter's canal.

FIG 48 Femoral arteriogram obtained on the asymptomatic leg the opposite leg was markedly involved. Note the large atheroma at the upper end of Hunter's canal.

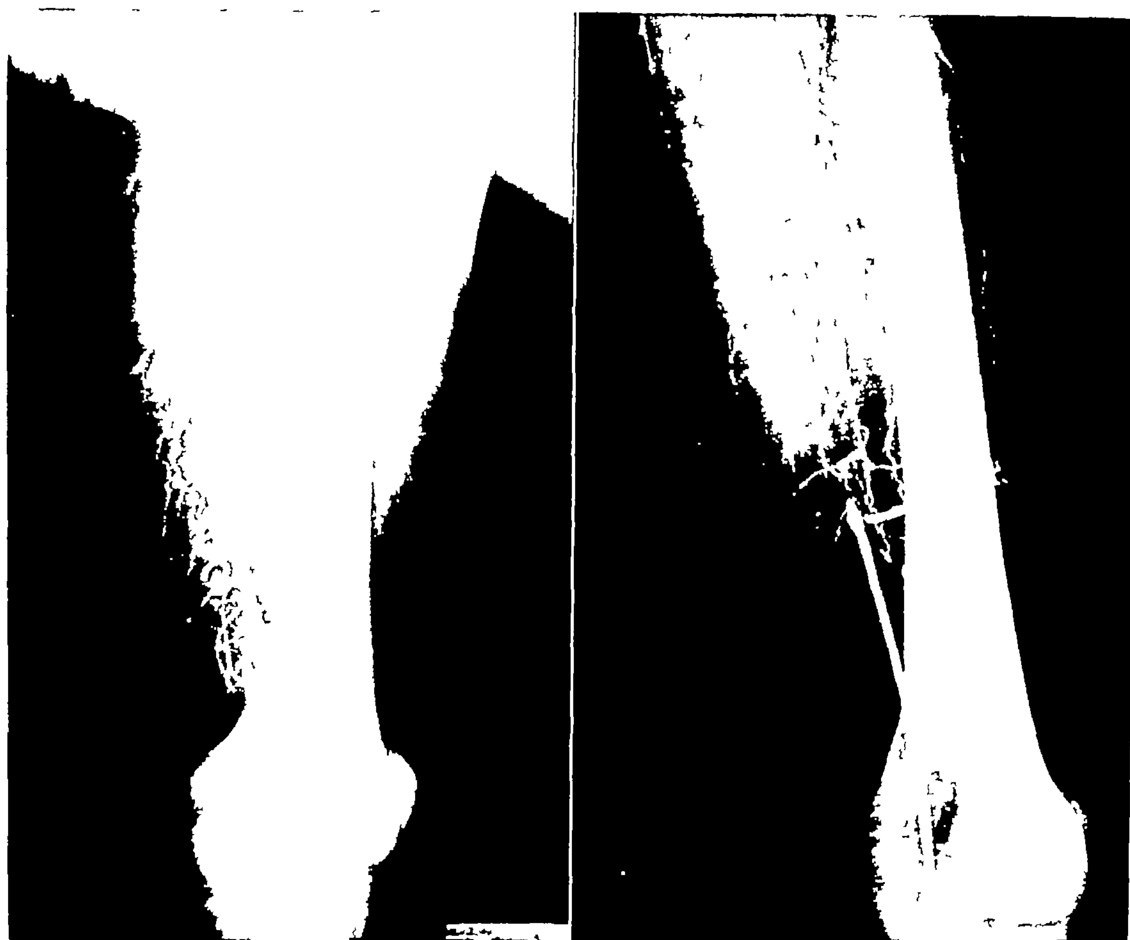


FIG 49 FIG 50

FIG 49 Short obstruction of the superficial femoral artery with sturdy collaterals A fair pulse was present at the ankle The bypass formed by nature is as good as any surgery could construct The contour of vessel above and below the obstruction is satisfactory

FIG 50 Bulbous dilatation at the distal end of an arterial obstruction This indicates a fair pressure in the segment distal to the occlusion The large collateral entering the distal segment must be preserved during surgery This is an ideal case for a femoral bypass

of Hunter's canal or a limited endarterectomy, may be considered when arteriograms are performed on the "good" leg Figure 49 shows a short obstruction of the superficial femoral artery at the upper end of Hunter's canal with a sturdy collateral and several others helping to shunt the flow Such a film would lead us to do a lumbar sympathectomy and *no direct restoration of continuity at this time*, since nature has done such a beautiful job Of course, if sudden obstruction should obliterate these collaterals, excision and grafting may become necessary In figure 50, the lower segment of the closed superficial femoral artery shows a small bulbous dilatation, a phenomenon one can observe when adequate collateral circulation through branches of the profunda maintain good blood flow and adequate patency of the distal segment

When the lower femoral or popliteal segment is not visualized in spite of clinical evidence of fair circulation below the knee, the femoropopliteal segment can be injected after surgical exposure At times, sufficient distal circulation is found to justify a bypass procedure (fig 51 A and B)



A B

FIG. 51 (A) The lower end of this 14 × 36 inch film has been cut off to save space It showed no refilling at the popliteal level The visible vessel in the thigh is the profunda (B) The lower femoral segment has been injected with 35 per cent Diodrast. It shows a fair-sized peroneal anastomosing with the posterior tibial artery The anterior tibial artery is absent.

Many correlations can be made between the clinical findings the oscil lometric curves and the angiograms The last must be interpreted in the light of other findings but the clinical observation is always the deciding factor because artefacts timing or spasm may give an erroneous angiographic pic ture With the advent of a direct attack on closed arterial segments however arteriograms have become indispensible for selecting appropriate surgical treatment

Phlebography

In spite of the extensive use of phlebograms in many of the continental and American clinics we have used this method with considerable reserve even although radiopaque solutions have been employed by us to demon strate unusual or undiagnosed venous obstructions for many years The reason for this attitude lies in the fact that (1) most cases of venous obstruc tion and venous insufficiency can be clearly evaluated by simple clinical tests, including venous pressure measurements (2) because of the larger cross section of the venous bed velocity of flow is diminished when compared with the arterial one and thus the danger of induced thrombosis is greater and (3) the interpretation of phlebograms is difficult and must be made with great caution

FEMORAL PHLEBOGRAM Injection of 35 per cent solution of Diodrast



FIG 52 Retrograde visualization of the femoral vein Note the filling of the venous cusps They retain some of the dye, but they do not hold under conditions of a rapid injection of a solution heavier than blood This does not mean a valvular incompetence

in the common femoral vein may be done in the horizontal or tilted, feet-down position to demonstrate the competence of valves If a postphlebitic syndrome is being investigated, one can readily place a cannula through the stump of the saphenous vein and demonstrate the location of valves and their insufficiency In figure 52 the solution has been injected through the sapheno-femoral junction The cusps of valves are well filled Each of the three valves retains some of the dye but they do not hold under the conditions of rapid injection under pressure As pointed out by Luke,[40] a number of "normal" deep venous segments exhibit such a reflux from above and do not necessarily show clinical signs and symptoms of incompetence

The femoral vein can also be visualized through a percutaneous injection into the short saphenous vein from the popliteal fossa Figure 53 demonstrates a comparatively high junction of the short saphenous with the femoro-popliteal segment The femoral vein shows a ragged contour and a few spotty

shadows characteristic of a thrombosed and recanalized vein Attention should be called to the fact however that flow conditions of the dye and various rates of mixing with blood can give pictures often difficult to interpret A less well canalized popliteal thrombus showing three distal channels and the origin of the anterior tibial vein from the tibioperoneal trunk is visualized in figure 54

An example of a well filling normal popliteal vein injected through a small varix in the short saphenous system is illustrated in figure 55 Two tortuous collaterals are visible above the knee emptying into the femoral vein The wealth of incompetent perforators overflowing from the deep veins to the two superficial systems is shown in figure 56 The clinical examination and the disposition of these cases to be discussed in chapter 13, indicate however that these films are mostly superfluous and their routine use has been vastly overdone Our clinic has always used them very sparingly They have not been used to localize incompetent perforator veins but more to demonstrate insufficiency of the popliteal vein



FIG 53



FIG 54

FIG 53 The dye has been injected percutaneously from the short saphenous vein. The lower femoral segment shows a ragged contour with shadows indicating mural thrombi. This is a recanalized femoral vein conducting blood in both directions depending on posture.

FIG 54 There is a subtotal occlusion of the popliteal vein at the level of the patella There are three sturdy veins distal to the occlusion. The origin of the anterior tibial vein from the tibioperoneal trunk is well visualized.





FIG 55 FIG 56

FIG 55 A normal poplıteofemoral venous segment ıs vısualızed through a varıx ın the short saphenous system A tortuous, probably ıncompetent, perforator veın empties ınto the femoral veın at the subsartorıal location

FIG 56 A large plexus of veins freely communıcates wıth the deep system from which the veins overflow The poplıteal veın ıs tortuous and unevenly filled Whether thıs ıs spastıc or organıc or due to uneven dıstrıbutıon of the dye ıs ımpossıble to tell It may be a recanalızed poplıteal segment

In many clınıcs, the short saphenous veın at the ankle ıs used to ınject and vısualıze the deep venous system, and thıs can be done ın the horızontal and standıng posıtıons, wıth or wıthout a blood pressure cuff used for venous congestıon Functıonal phlebograms, taken before and after exercıse, gıve ınterestıng and often confusıng pıctures In our clınıc, they have not found any use

Other venous segments requıre occasıonal vısualızatıon In compressıons or obstructıons of the superıor vena cava, angıograms readıly show an ıntrınsıc or extrınsıc lesıon (fig 57) The ılıac veıns can be readıly ınjected wıth 50 per cent Dıodrast, rapıdly, usıng 20 to 25 cc (fig 58) Note the wıde channel and even contour of the external and common ılıac veıns, the ınternal ılıac veın, after a tortuous course, ıs seen to empty at the posteromedıal wall of the trunk, producıng a bulbous dılatatıon A fine presacral and vertebral network ıs also vısıble

Intraosseous ınjectıons ınto the os pubıs, the ıschıum or trochanter have been suggested to vısualıze the parıetal and vısceral branches of the ınternal ılıac veın [41] These ınjectıons are dıfficult A sıngle experıence wıth ınjectıng 20 cc of 70 per cent Dıodrast outsıde of the pubıc bone sımply served to



FIG 57 Visualization of the subclavian and innominate vein together with the superior vena cava in the case of a mediastinal mass suspected of being an aneurysm It was due to Hodgkin's disease. No intrinsic lesion of the superior vena cava is seen



FIG 58 Visualization of the common iliac vein and its branches through a cannula inserted into the saphenofemoral junction. The patient had a congenital absence of valves in the deep venous system and showed massive reflux from above on standing. Note the fine pattern of prevertebral veins which enlarge in case of obstruction of the main channel.



FIG 59 Extravasation of 70 per cent Diodrast outside the pubic bone The dye disappeared in 24 hours

illustrate how rapidly the dye, even in this concentration, disappears (fig 59), and how comparatively safe such extravasation is compared with inorganic iodide or Thorotrast solutions Possibly gynecologists, by injecting the bones of the pelvis or the body of the uterus, will get a great deal of information from this source

We have had more experience with the injections into spinous processes to visualize the vertebral veins and the vena cava (fig 60) The fine arcuate network gathers into an anterior and posterior vertebral vein, two lumbar veins are also well visualized A much coarser pattern, together with pooling and blockage of a distended lumbar vein, is seen in figure 61 The anterior and posterior vertebral veins are hardly or spottily visualized This patient developed a paraplegia following an endarterectomy of the aorta, and a tentative diagnosis of thrombosis of the vena cava and vertebral veins was made, a syndrome which Coelho and I recently described [42]

There are, of course, many other situations which may be illuminated by phlebography Generally speaking, the injections should be done in three to five seconds, seldom with a tourniquet, and the opaque solution should not be heavier than 35 per cent, unless the iliacs or the vena cava are to be visualized With such precautions and with preliminary intravenous testing for iodine sensitivity, untoward reactions can be kept at a minimum



FIG 60



FIG 61

FIG 60 Visualization of the vertebral veins through injection of 70 per cent Diodrast solution into the spinous process of a lumbar vertebra. Note the fine arcuate veins gathering into an anterior and posterior vertebral vein Two lumbar veins are well seen.

FIG 61 The pattern of the vertebral veins is much coarser than in the previous film A lumbar vein coursing anteriorly is blocked. This patient had evidence of vena cava thrombosis, but also had evidence of a sudden paraplegia, which was difficult to explain on an arterial basis.

Aortography

DIRECT LUMBAR ROUTE Puncture of the abdominal aorta with a 6-inch 17 gauge needle and a rapid injection of 70 per cent Diodrast or 75 per cent Neo-Iopax in quantities of 20 to 40 cc visualizes the renal circulation the celiac axis, the superior and inferior mesenteric arteries the bifurcation of the aorta, the iliac arteries and also the femorals (fig 62) If the film is 14 by 34 inches long and the tube distance is 48 to 52 inches away from the cassette the vascular tree can be visualized as far as the popliteal segment The patient is premedicated with 200 mg of Luminal sodium an hour before the injection and 100 mg of Demerol hydrochloride are given intravenously just before the aortogram. He has been tested the day before with an intra venous injection of 1 cc. of the organic iodide solution to be used The patient lies prone on the cassette and the needle is inserted proximally 7 cm. a handwidth, left of the midline just below the twelfth rib (fig 63) The needle is elevated cephalad and medially it touches the body at the first lumbar vertebra and is then redirected anteriorly skirting the body of the

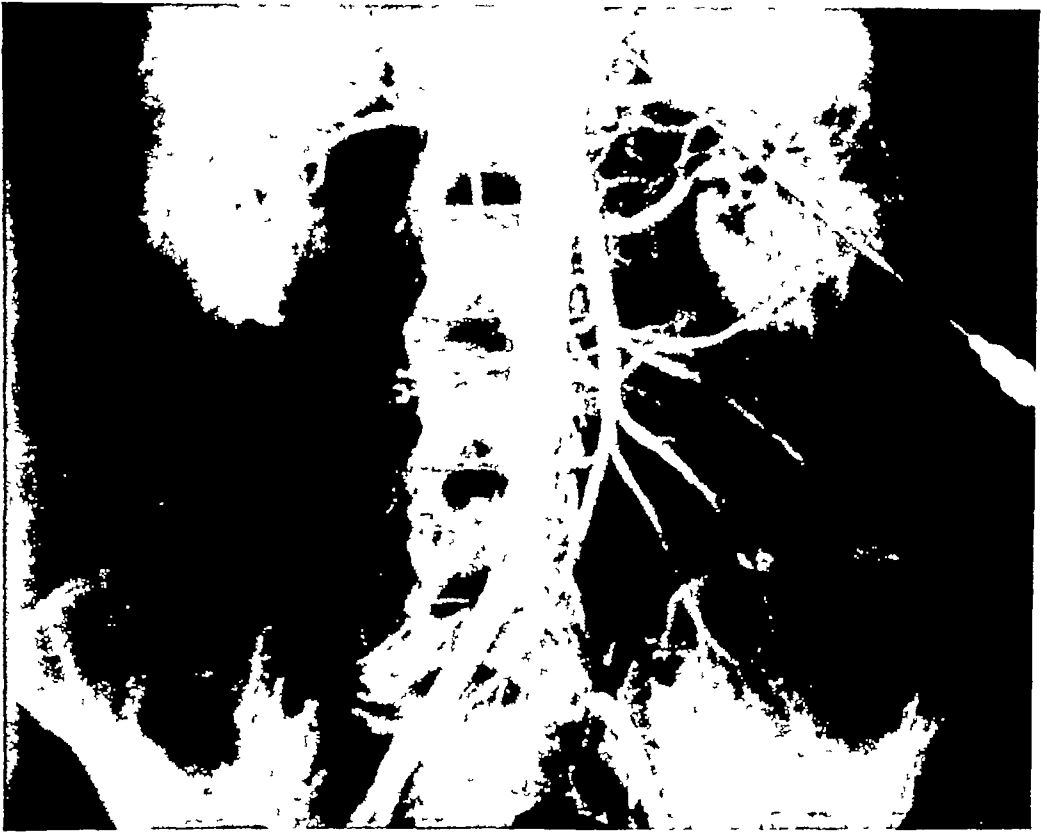


FIG 62 The celiac axis, a well visualized superior mesenteric, the inferior mesenteric and some lumbar arteries are seen The aorta is moth-eaten and narrow below the inferior mesenteric artery The right common iliac is sturdy but judging from its changing density, there is a plaque at its bifurcation into the external and internal iliac arteries On the left there is severe destruction to the common iliac and internal iliac arteries The patient had left-sided gluteal claudication

vertebra As it approaches the aorta, pulsation is transmitted to the needle, and after the aorta is entered arterial blood is seen to appear in the polythene tubing There is seldom pulsatile flow because of the length and the comparatively narrow diameter of the needle

The roentgen technician is now alerted by counting one, two and three, and at the command "shoot" he exposes the film By this time the opaque substance has been emptied from the syringe, preferably in two seconds There should be a few cubic centimeters left in the syringe when the picture is taken This is the simplest form of aortography done by hand and with one exposure

On such a film (fig 64) one watches for the diameter and contour of the aorta Note the gradual narrowing of the abdominal aorta starting at the level of the renal arteries There seems to be an atheromatous plaque just below the right renal artery Stenosis of the renal artery is not present The lumbar arteries can be seen Attention is focused on the bifurcation of the aorta which seems to contain plaques, both iliacs are narrow and irregular Both hypogastric arteries fill, but the density of the shadows is uneven This patient has slightly diminished femoral pulses and vague gluteal and thigh cramping on walking He is 47 years old and his father died of generalized arteriosclerosis in his fifties

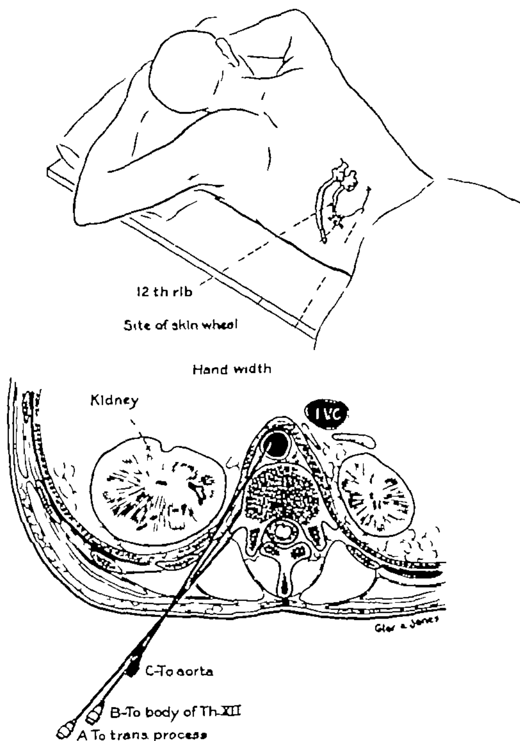


FIG 63 Technique of lumbar aortography Patient lies prone on the cassette The needle is inserted a handwidth left of the midline, slightly below the tip of the twelfth rib This, however is not always palpable. The tip of the needle is raised cephalad and medially it should hit the body of the first lumbar perhaps the twelfth dorsal vertebra. Then it skirts the lateral wall of the vertebra until transmitted pulsation is encountered. The anesthetist is now alerted, who starts the Pentothal anesthesia. The needle enters the aorta. The dye is injected rapidly and the film obtained while a few cubic centimeters are still in the syringe.

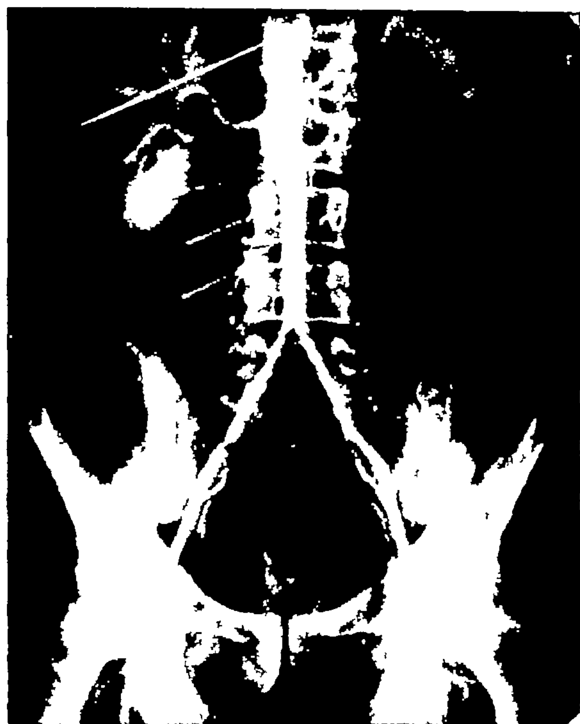




FIG 64 FIG 65

FIG 64 The abdominal aorta becomes narrower just below the right renal artery At the bifurcation the contour is ragged and the common iliacs show mottling, which is usually due to plaques Both common iliacs are narrow Both hypogastric arteries fill, but the density of the shadows is uneven

FIG 65 Huge atheroma of the aorta just above the bifurcation The two renal arteries and the splenic artery are well visualized The iliacs are narrow and irregular, the right is worse than the left, which was consistent with the clinical findings This is an inadvisably low aortic puncture, and injection into the superior mesenteric or renal artery may occur While the aortogram is informative, this angle of injection is faulty

In figure 65 a huge atheroma is visualized just above the bifurcation, together with small irregular iliac vessels The insertion of the needle here is unnecessarily low and is more conducive to cord damage

Various pressure tanks have been devised to deliver the pressure at an even rate with even force A foot pump can be used, as in the Bird-Campbell tourniquets Also, multiple exposures can be taken, preferably three at two second intervals, thus giving a more adequate idea of the type and extent of the circulation distal to the obstruction To date we have been quite successful in obtaining the necessary information by using a hand-operated syringe and a single exposure, although there is a great deal of room for improvement Serial aortograms and arteriograms will undoubtedly be universally used in the future.

SIDE EFFECTS AND HAZARDS The question of anesthesia has not been settled. At St Luke's Hospital, Pentothal sodium with oxygen is administered in the prone position The patient is not intubated and the procedure is not without risk The patients, however, like this method

At the University of Illinois the procedure is done under local anesthesia In spite of adequate premedication there is diffuse visceral pain on entering the aorta, and after the opaque substance is injected, pain may radiate up and

down along the course of the aorta Both anginal and deep pelvic pain have been observed

The immediate cardiovascular response to the aortogram has been carefully studied by Deterling [43] In the experimental animal and in man there seems to be a constant vasomotor pattern Following a short transient rise of blood pressure there is a period of hypotension with bradycardia and an occasional ventricular extrasystole Finally there is hypertension with tachycardia which lasts from one to five minutes Diodrast gives less of a reaction than Neo-Iopax our service has never used Thorotrast or sodium iodide because the former deposits a radioactive substance which is detectable with a Geiger counter many years after injection and the latter is highly irritant to endothelium and evokes intensive vasomotor response Urokon is favored by some roentgenologists, and so is Hypaque

Mesenteric thrombosis death and paraplegia have been reported following lumbar aortography for which reason it should never be undertaken without definite indications [44] In a patient whose femoral pulses are absent or weak who has claudication in the buttocks and thighs and who shows little or no trophic changes in the feet the diagnosis of aortic or common iliac stenosis or occlusion is certain Aortography delineates the extent of the



FIG. 66 Dissecting injection of dye, probably entering a line of cleavage between atheroma and medial fibers. The dye spreads cephalad and ends abruptly showing no branching. The dye disappears in 24 hours. Two such injections have been seen both created days of discomfort and some reflex ileus.

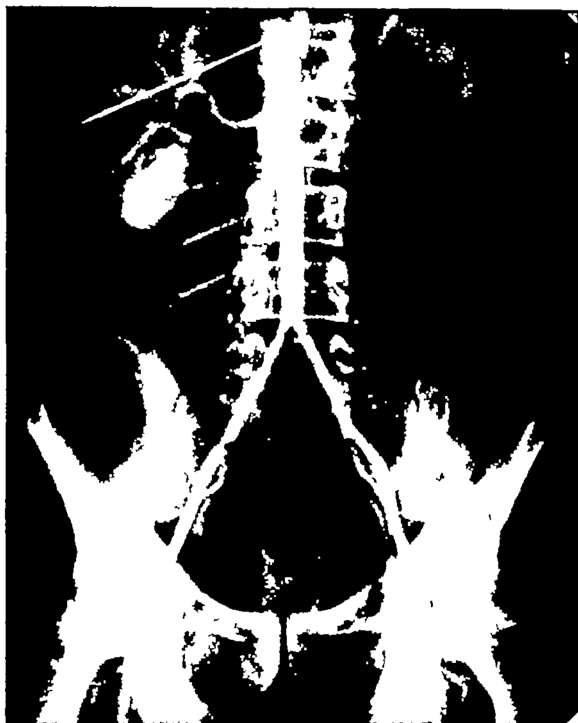




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At the University of Illinois the procedure is done under local anesthesia In spite of adequate premedication there is diffuse visceral pain on entering the aorta, and after the opaque substance is injected, pain may radiate up and

down along the course of the aorta. Both anginal and deep pelvic pain have been observed

The immediate cardiovascular response to the aortogram has been carefully studied by Deterling.[43] In the experimental animal and in man there seems to be a constant vasomotor pattern Following a short transient rise of blood pressure there is a period of hypotension with bradycardia and an occasional ventricular extrasystole Finally there is hypertension with tachycardia which lasts from one to five minutes Diodrast gives less of a reaction than Neo-Iopax our service has never used Thorotrast or sodium iodide because the former deposits a radioactive substance which is detectable with a Geiger counter many years after injection, and the latter is highly irritant to endothelium and evokes intensive vasomotor response Urokon is favored by some roentgenologists and so is Hypaque

Mesenteric thrombosis death and paraplegia have been reported following lumbar aortography for which reason it should never be undertaken without definite indications [44] In a patient whose femoral pulses are absent or weak, who has claudication in the buttocks and thighs and who shows little or no trophic changes in the feet the diagnosis of aortic or common iliac stenosis or occlusion is certain Aortography delineates the extent of the



FIG 66. Dissecting injection of dye, probably entering a line of cleavage between atheroma and medial fibers. The dye spreads cephalad and ends abruptly showing no branching. The dye disappears in 24 hours. Two such injections have been seen both created days of discomfort and some reflex ileus.

occlusion and helps to determine, although never decides on, operability It need not be done in abdominal aneurysms at all [44]

The most important safeguard against mishaps is the *constancy of personnel* performing the procedure The rotating surgical resident should be supported and checked by a younger member of the permanent staff. This way blank injections or periaortic dissecting deposits will be kept to a minimum When organic iodides are deposited outside of the vessel, the pain seldom lasts more than a few hours The solution occasionally is injected into the wall of the aorta, producing a weird shadow which one quickly recognizes as being due to dissection of the opaque substance (fig 66). No branches are visualized in such a dissecting type of injection The solution disappears in a few hours

RETROGRADE AORTOGRAPHY Personal experience is limited to a few femoral exposures through which a polythene catheter is threaded and the solution injected against the stream at a desired level With a single exposure one often obtains a film like that shown in figure 67, indicating a complete occlusion of the aorta but giving no information regarding the state of the iliac vessels These might appear several seconds later in serial films, or may be shown by retrograde injections through both femoral arteries Injection through the left femoral artery of the same patient (fig 68) indicates a good external iliac artery, with a badly damaged left common iliac and a partially



FIG 67



FIG 68

FIG 67 Total occlusion of the abdominal aorta, showing a filling of both renals and a huge atheroma just below the left renal No filling of iliac vessels is seen A serial injection here may have revealed retrograde filling

FIG 68 Retrograde visualization of the aorta through injection of the left femoral artery The left external and internal iliac vessels fill well but the left common iliac is badly damaged The aorta shows a partially open channel to the upper level of the third lumbar vertebrae The right common iliac artery has filled, perhaps through presacral anastomoses

occluded aortic channel to the upper end of the third lumbar vertebra right common iliac also filled partially from this injection When the inje was made through the right femoral artery an excellent filling of the common external and internal iliac arteries was obtained (fig. 69) Rese of the aortic bifurcation has a much better chance of succeeding whe distal segments are only slightly involved However the puncture sclerotic femoral artery and the insertion of a catheter through it are hazardous than the injection of the needle in a rapid flow of blood i abdominal aorta The only indication for retrograde aortography is the tionable state of external iliac and femoral segments when translu aortography has failed to adequately visualize them

The thoracic aorta can also be visualized either by direct punctu retrograde through the ulnar or carotid arteries The description of methods is beyond the scope of this volume but has been critically disc by Deterling, W G Scott and others [43] The technique of Wilborn h cently been employed by our cardiovascular unit[45] (fig 33)

A combination of aortography with presacral injection of air may s times visualize an adrenal tumor In figure 70 the slightly elongated tor aorta with two normal renal arteries is shown The outline of the kidn

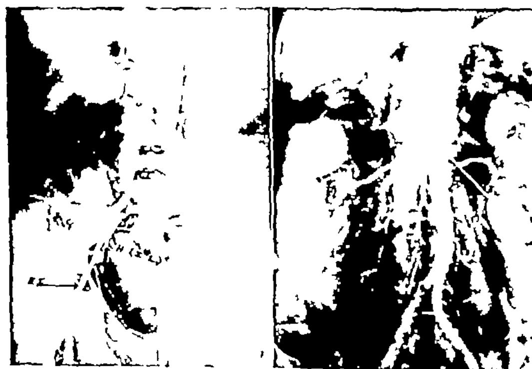


FIG 69 FIG 70

FIG 69 Retrograde femoral arteriogram of same patient whose film is shown in fig The entire tree of the common iliac, external iliac and internal iliac arteries is patent. are blocked at the aortic bifurcation.

FIG 70 Normal aortogram in a patient suffering from essential hypertension. pheochromocytoma was suspected, the aortogram was done following presacral infilt of air Both renal arteries are well seen. There are no abnormal shadows in the adrenal r This was verified later during splanchnicectomy The lower end of the aorta and the le artery are slightly tortuous due to the hypertension

unusually sharp, the two adrenals are doubtfully visualized at the level of the first lumbar vertebra, but no enlargement can be seen. The patient had essential hypertension and during splanchnicectomy both adrenals were found to be normal

5. METHODS OF CLINICAL RESEARCH

Constant Temperature Room

Neither readings of skin temperature nor plethysmographic curves can have anything but comparative values unless they are carried out in a constant environment There are inexpensive, almost portable, apparatuses and there are elaborate machines with baffles or perforated ceilings to avoid drafts Humidity control is more important in hot than in cold environmental temperatures. It should be possible to accomplish rapid cooling of the room to 60° F (20° C) and rapid warming to 90° F (32 2° C). Reflex release of vasomotor tone can be usually complete at 82° F (28° C) If only one control temperature is readily possible studies are made at 75° F (24° C) with 50 per cent humidity, the so-called "comfortable room"

Outside noises, drafts and unfamiliar and formidable looking instruments all influence the measurements, especially when sensitive plethysmographs are used for study The individual should be kept in a constant environment of temperature and humidity for 60 minutes before determinations are started, and by this time he is either well relaxed or asleep or fit to be tied These facts are mentioned not to decry the use of this equipment but to emphasize how careful, painstaking, expensive and cumbersome such a study is in order to be meaningful

A factor which makes cooperative studies difficult between centers of different locations is that the vascular responses are different in patients living in a cold and dry climate from those living in a hot and humid climate This was observed in a study undertaken for the National Research Council by five cooperating centers * Aside from carefully controlled individual research, there seems to be no need for governmental or private institutions to invest in a constant temperature room, especially if there is no full time personnel to go with it Vascular surgical services can function well without it

Sweating Tests

There is growing evidence that vasomotor impulses and the sudomotor outflow do not necessarily correlate with each other While absence of sweating, as observed clinically with the palpating hand, is customarily used to test for the extent of denervation, the possibility of anatomical dissociation of vasomotor and sudomotor pathways is quite suggestive (H B Hertzman, et al.)[46] Nevertheless, for practical purposes one may assume that the nonsweating skin has lost its vasomotor control

*Cold Injury Project, No 175 Committee on Veterans' Medical Problems, National Research Council Drs Elkin, Burch, Glenn, Simeone and de Takats

Sweating may be studied by the naked eye by using a magnifying lens to study the number of sweat drops by the application of starch iodine Quinizarine or cobaltous chloride Since our experience has been mostly with the starch iodide method of Minor[47] this will be described in detail A mixture is prepared containing iodine 1 52 gm. castor oil 10 cc and absolute alcohol to make a total volume of 100 cc. The skin must be completely dry and clean before the mixture is applied The solution is nonirritant but of course patients with sensitivity to iodine are eliminated The eyelids and external genitalia are avoided When the solution dries fine rice starch powder is dusted on it with a powder puff and all excess fanned away Two large electric bakers are now placed over the patient Further increase in sweating may be accomplished by giving 15 gr (1 Gm) of aspirin half an hour before the test and several cups of hot tea Pilocarpine is not used Sweat will first appear in fine dark blue dots resembling poppy seeds, which



FIG 71 This 57 year old railroad engineer was studied because of postural hypotension. While he had no Horner's syndrome he acted as if the *four upper dorsal sympathetic ganglia* had been removed. He showed no sweating on the starch iodine test above the nipple line.

finally coalesce and form a violet-black surface This can be readily photographed for a permanent record (fig 71) The mixture can be easily removed with soap and water

A rapid simple method of testing for electrical skin resistance can be used to study the extent of sympathetic denervation A new portable instrument has been described by Whelan [48] The skin resistance is essentially due to the activity of sweat glands and the method is so sensitive that it can be used advantageously to determine early regeneration of the sympathetic nerves after interruption The skin resistance is not only high after sympathectomy, but it does not fluctuate since it is not influenced by the multitude of stimuli which normally excite sudomotor and vasomotor activity Felder, et al ,[49] found that the returns of sudomotor and vasomotor activity are usually concomitant, even though these modalities are conducted by different pathways (fig 41)

Sweat glands remain intact in the sympathectomized skin[50] and both pilocarpine and Mecholyl chloride have a peripheral effect on sweating by stimulating chiefly the cholinergic nerve fibers.[51] All types of sweating, namely the thermo-regulatory, emotional, drug-induced and axon reflex, are abolished by sympathectomy [52]

Plethysmography

Many instruments have been devised for recording volume changes of a

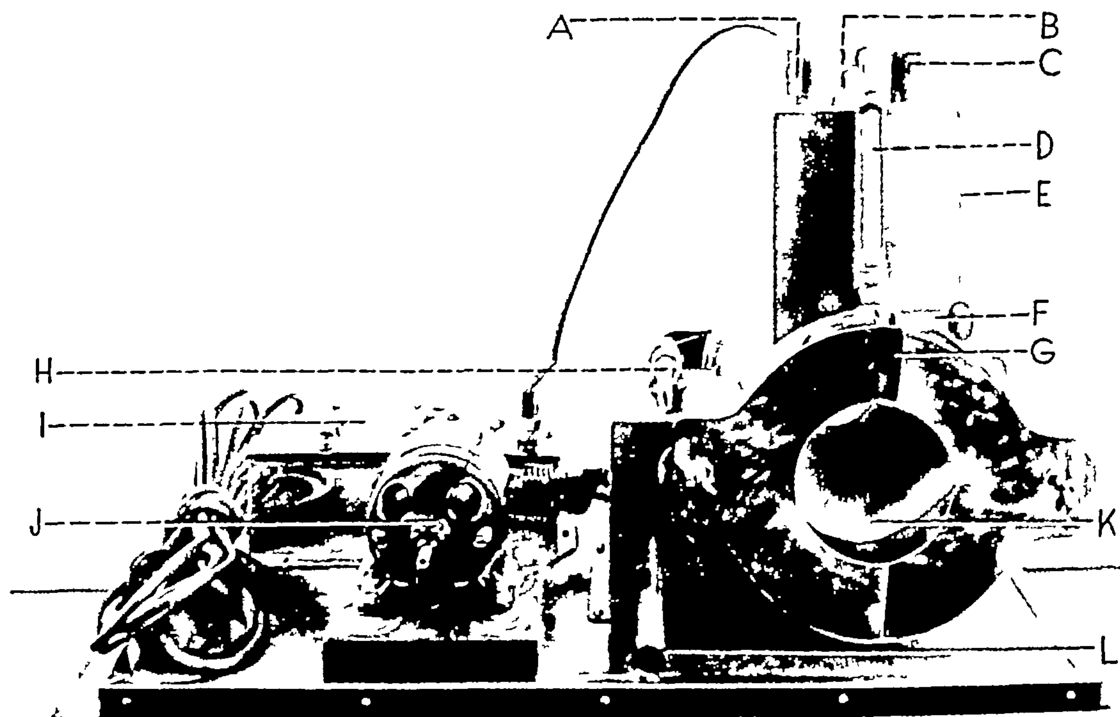


FIG 72 Final design of plethysmograph, the result of several modifications (A) Insertion for electroregulator (B) Outlet leading to kymograph (C) Outlet for thermometer (D) Indicator of water level in plethysmograph (E) Cubicle for foot (F) Thumbscrew to tighten rubber dam (G) Metal panel encircling hand or foot (H) Plunger to calibrate blood flow after each determination (I) Switchbox (J) Motor (K) Suspension for extremity (L) Outlet (Miller and de Takats Posttraumatic Dystrophy of Extremities Surg Gynec and Obst, 75 558, 1942)

part since the initial publication of Brodie (1905) who enclosed an organ such as the kidney in an oncometer and then obstructed the venous outflow while recording the rate of swelling of the part as blood accumulated in it This principle was adapted to the human arm by Howlett and Van Zwaluven burgh in 1909 they enclosed the arm in a chamber and obstructed the venous outflow with a blood pressure apparatus by inflating it to a pressure below the diastolic pressure Thus the *venous occlusive plethysmograph* measures the volume increase of the arm from which the rate of flow can be readily calculated As a graduate student Donald S Miller modified the Abraham son Ferris apparatus [53] and many hundred blood flow determinations were made at the University of Illinois in a study of reflex sympathetic dystrophy in 1942[35] (fig. 72)

Many plethysmographs have since been developed for arms legs and especially for digits (White Smithwick and Simeone[32]) Burch and Winsor

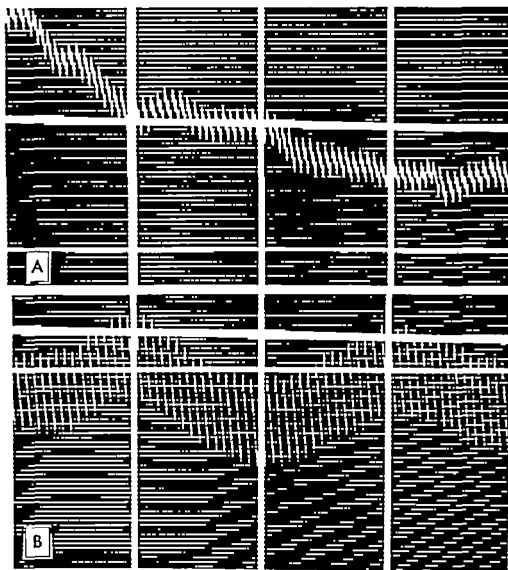


FIG 73 Tracings obtained from right middle finger with a Burch-Winsor plethysmograph (A) before and (B) 10 minutes after an intra-arterial injection of ½ gr (0 03 Gm.) papaverine

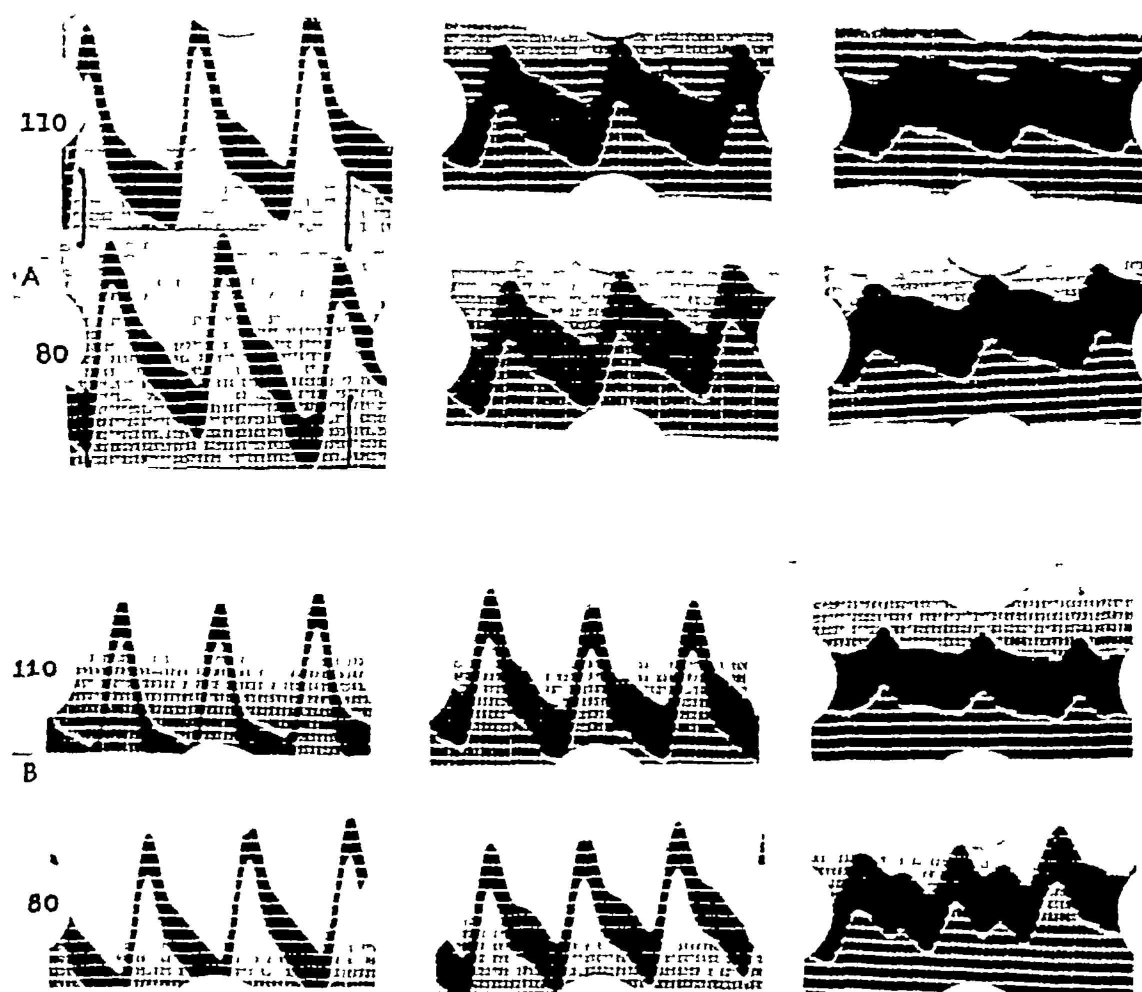


FIG 74 Pulse wave recorder (recording oscillometer) of Carl Johnson The three columns from left to right represent recordings at midthigh, just above the knee and below the knee (A) Prior to operation, indicating huge pulse waves with sharp peaks above and diminished pulse waves with blunt peaks below a stenosis of the superficial femoral artery at Hunter's canal (B) Three months after endarterectomy, the pulse waves above the obstruction are lower and below the obstruction are higher and sharper than preoperatively The graph well depicts the hemodynamics of arterial stenosis (de Takats, G Clinical and Angiographic Correlations in Arterial Stenosis. J A M A , *158* 1502, 1955)

have devised a digital plethysmograph, with which we made many observations on war veterans who suffered arterial or cold injuries * This is a closed pneumatic system which includes the tip of the finger or toe. Changes in the volume of the enclosed part are transmitted by the pneumatic system to a bellows-type diaphragm which moves a string and which in turn is photographed by a light and lens system This plethysmograph is thus a volume recorder operated on a pressure of less than 1 to 2 mm of water [54] From the amplitude of the pulse waves, blood flows are calculated in cubic centimeters per 5 Gm of tissue (fig 73).

A sensitive, ink writing instrument has been recently described by Simeone and his co-workers; it can be used as an oscillometer to record the pulsation at any desired level of the extremity and also as a digital plethysmograph [55]

* Cold Injury Projects No 14, 17 and 175 Committee on Veterans' Medical Problems, National Research Council

One fundamental objection to all plethysmographs is the fact that volume pulsations of a limb or digit do not necessarily correlate with blood flow and that distention and pressure in the veins greatly influence the pulse wave. An alteration of venous pressure as accomplished by different positions of the leg, changes pulsations measured by the plethysmograph. Raising the part and emptying the veins usually results in apparently increased flow.[56] In addition to changes in the venous reservoir reflex constriction and dilatation of the terminal arteriocapillary bed may influence the record. This is the reason why efforts to calculate blood flow from an otherwise excellent pulse wave recorder such as Johnson's,[57] have not been regarded as convincing (fig 74). However for a permanent record such an oscillometer is superior to a manual one as discussed on page 49.

Stethogram

The visual recording of murmurs or bruits, which are so important in the localization of aneurysms and arterial stenoses, can be readily accom-









FIG 75 Stethogram above and below the upper level of a saddle thrombus of the aorta. The recorded murmur is highest 2 inches above the navel, and absent 1 inch below the navel. Aortogram and laparotomy confirmed the exact location of the obstruction.

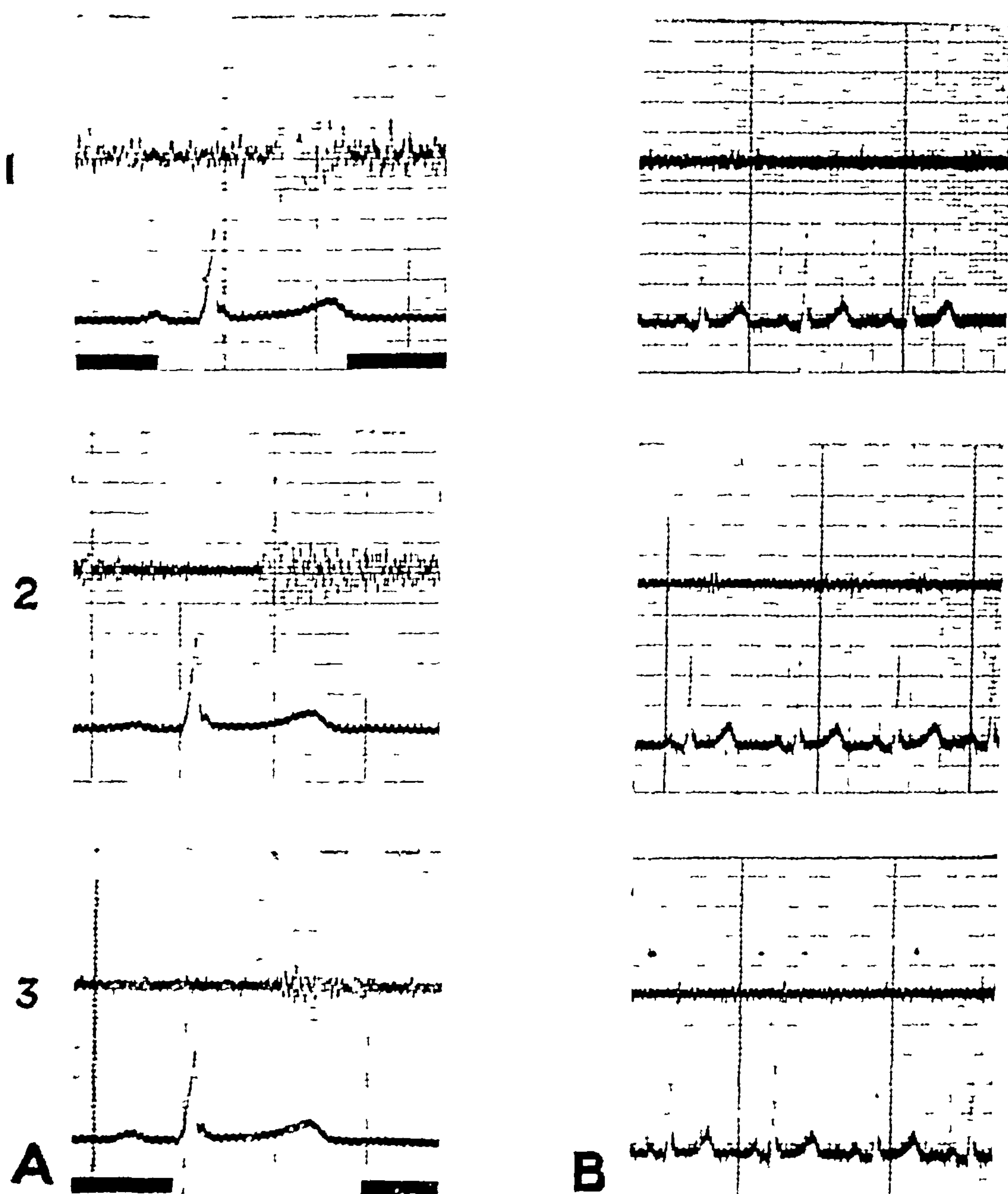


FIG 76 Stethograms indicating the loudest murmur at (1) in the middle of the left upper arm, where a congenital arteriovenous fistula was located At (2) and (3), distal to this point, the murmur was less intense Following closure of this fistula at (B), no murmur was audible or could be recorded

plished by a simple device which is attached to an electrocardiograph Figure 75 illustrates the bruit which was audible in the case of a saddle thrombus of the aorta and which disappeared 1 inch below the navel, exactly where the obstruction began In figure 76, the left three records are taken from the right upper extremity of a 12 year old girl who has had six previous operations for multiple congenital arteriovenous fistulae The loudest and highest of the murmurs was heard at (1) in the middle of the upper arm, and this is where the tracing shows the highest amplitude Shortly following resection of this communication, no bruit was audible and the tracing at (B) shows that the bruit had disappeared

For many years we have used a stethogram for the localization of arterial stenoses and arteriovenous fistulae In addition to the loudness of the bruit the highest amplitude corresponding to the highest tones of the murmur on auscultation are excellent localizing signs of the fistula However a good stethoscope leading to a good ear makes the routine use of a stethogram unnecessary

Capillary Microscope

A binocular microscope magnifying 50 to 100 times and illuminated with a filtered light visualizes manifold capillaries and may detect the number of capillaries per unit area their configuration and the speed of the red corpuscles flowing through them The method has been extensively used in the past and photographic and cinematographic attachments have been added At our vascular clinic photomicrographs have been taken through the courtesy of Dr A Nedzel of the Department of Pathology at the University of Illinois It cannot be said that definite diagnostic or therapeutic assistance has been rendered by this method with the possible exception of the study of conjunctival vessels when exposed to cold saline solution Certain patients suffering from Raynaud s phenomenon seem to show sludge and agglutination of red cells [58] Such studies are readily carried out in the Department of Ophthalmology at the University of Illinois and we have made occasional use of them

Ergometry

Walking tests and treadmills have been utilized by many to permit a quantitative estimate of the performance of an ischemic extremity compared when feasible to that of a normal one Actually the patient s own estimate of his limitation *i e.*, how far he can walk at a normal rate without cramping of the muscles, is a rough type of ergometry but a very practical and useful one When the performance of both limbs is tested simultaneously such as on a treadmill it is impossible to separate the part that an individual limb plays in the total effort The ergometrograph of Hitzrot and Naide[59] records muscle action on electrical stimulation and is slightly uncomfortable Simeone described an apparatus based on the Mosso principle but with the major modification that the exercise develops a progressively increasing resistance against which the muscle has to work. The total duration of the exercise the pressure developed and the time elapsed before the appearance of fatigue or of muscle cramping are taken as indices of ergometric performance The method has been used by Simeone in the late follow up studies of war veterans suffering arterial injuries * He found a strong correlation between ergometric performance and the rise of digital temperature following posterior tibial block in a cool room Interestingly the perform

*Cold Injury Project No 175 Committee on Veterans Medical Problems, National Research Council. (To be published.)

ance of upper extremities after arterial wounds was relatively more depressed than the performance of the lower extremities, even although collateral circulation is notoriously more abundant in the upper than in the lower extremities Ergometry is, of course, a test of muscular performance which may be influenced not only by deficient circulation but by nerve involvement and by actual disease of muscle Its greatest drawback is that a subjective element cannot be eliminated The experience of our group with this apparatus has been quite limited Recently, however, a myoergograph, which registers individual tetanic contractions and also a fatigue curve obtained by repeated contractions, has been investigated The calf muscle is stimulated by a rhythmic oscillating current and does not depend upon the patient's volition [60] Personal experience with this apparatus is just accumulating.

Circulation Times

The classic methods of using dyes such as Decholin, sodium cyanide, magnesium sulphate, ether or Solu-B, which signal their arrival by taste or smell, are of more interest to the cardiologist than to the vascular surgeon Considerable interest has been shown, however, in venous circulation times measured from foot to carotid at bed rest, on elevation or following operations They were definitely prolonged after operation Histamine flares can be raised in certain areas of the skin and then fluorescin injected intravenously The wheal begins to fluoresce when illuminated with an ultra violet lamp fitted with a Corning filter [61] To all such circulation times one can raise a series of objections The rate of injection and the concentration of the injected substance together with the detectable amount at arrival are all variable Furthermore, the circulation time between two points is equal to the volume of blood contained in the vessels between the two points divided by the flow [62] Thus, during vasoconstriction, which decreases volume, flow might also decrease and not actually affect circulation time appreciably When catheters are used, vasoconstriction often occurs With the advent of radioactive isotopes, most of these methods have been superseded

Clearance Methods

For measurement of peripheral flow, the clearance of radioactive sodium or iodine from the tissues injected has been utilized by Elkin and his associates [63] A small amount of the radioactive ion is injected and the rate of clearance from the area is followed by a counting device This again is an indirect measure of blood flow, and the permeability of the tissue-blood barrier is also involved When such a method is compared with skin temperatures or plethysmography, striking discrepancies appear Arteriovenous shunts may play an uncontrollable role Because of the expensive and complicated setup, we have not been convinced that these methods will greatly add to the understanding and evaluation of circulatory disturbances These studies did emphasize, however, that selective regional vasodilation, not

general vasodilation should be the object in the treatment of vascular disease

Radioactive iodine can also be injected into the long saphenous vein at the ankle and the arrival of the tagged albumin is picked up at the groin with a scintillating counter With this method it could be shown that by the application of an elastic bandage to a lower limb elevated three inches above the horizontal the greatest decrease in circulation times could be obtained [64]

Blood Gas Studies

We have obtained data in the middle 1930 s of arteriovenous differences of oxygen and carbon dioxide and of venous saturation of oxygen under conditions of rest exercise intermittent venous hyperemia and sympathetic block * With the knowledge since accumulated on the arteriovenous shunts many of these studies require reinterpretation The degree of oxygen saturation of arterial blood may be expressed as

$$\frac{\text{Oxygen content}}{\text{Oxygen capacity}} = 93 \text{ per cent}$$

The oxygen content of normal arterial blood has been estimated to have an average value of 18 5 volumes per cent and that of the venous blood 15 volumes per cent The coefficient of oxygen utilization by the tissues is the result of many factors and is determined as follows

$$\frac{\text{arterial oxygen} - \text{venous oxygen}}{\text{arterial oxygen}} = \frac{18.5 - 15}{18.5} = \frac{3.5}{18.5} = 18.9 \text{ per cent}$$

This figure varies with the metabolic activity of the tissues the oxygen capacity and the hemoglobin saturation the rate and volume of blood flow and the efficiency of the capillary exchange Because of the many variables it does not seem likely that measurements of cerebral or peripheral blood flow can be deduced from any of these figures Thus the cerebral A V oxygen difference, being the product of the interrelation between cerebral blood flow and cerebral metabolism cannot of itself be a measure of either [65] For this reason, Kety applied the Fick principle to the measurement of cerebral blood flow [66] In its simplest form this principle states that the quantity of a given substance taken up by an organ from the arterial blood equals the amount of the substance carried to the organ by the arterial blood minus the amount *removed by the venous blood during the same time* or $Q = QA - AV$ Q is the total quantity of the substance, A and V are its content in arterial and venous blood respectively Chemically inert gases (nitrous oxide and radioactive krypton) have been used for such a substance

In this test it is not the absolute flow but the flow per 100 ml of metabolically active tissue which is measured. If some tissue such as a cerebral infarct, is without blood flow it is also inactive in taking up the foreign substance, and thus changes in blood flow following sympathetic block for apoplexy will not be registered [67] For the peripheral circulation the Fick

* de Takats, Hick and Kendrick, 1935. (Unpublished.)

principle is not applicable because the abundance of venous collateral network interferes with obtaining a representative venous sample

Many years of study on the polarigraphic study of oxygen tension in the tissues has been recently summarized by Hugh Montgomery [68] The potentialities of this method are great, but its clinical usefulness awaits further standardization and simplification

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PART III

Vascular Syndromes Requiring Surgical Care

It is the customary fate of new truths to begin as heresies and to end as superstitions

THOMAS HENRY HUXLEY 1825–1895

CHAPTER 7

CONGENITAL VASCULAR ANOMALIES

Definition

In view of the complicated network of vessels from which the final if not always standard pattern of the vascular tree evolves it is a wonder as Mont Reid remarked that direct communications between large arteries and veins do not occur more frequently [1] Before large arterial and venous channels develop in any part of the body a diffuse capillary plexus is present which is formed from the mesenchyme from this plexus the arteries and veins are differentiated by the enlargement of certain channels and by the atrophy and obliteration of others *Anomalous blood vessels occur when unusual paths are chosen in the primitive vascular network for the development of the permanent pattern when normally obliterating vessels persist when normally retained vessels disappear and when normally distinct channels fuse or incompletely absorb*

Classification

Woollard [3] in a classic study of the development of the principal arterial stems described three well defined stages the plexiform stage the retiform stage and the stage of stem formation It is easy to see that the varied clinical picture of vascular malformations is due to the arrest or modification of development of the normal process Thus in previous communications it was pointed out[4] that one can correlate the well defined stages of Woollard with clinical pictures of vascular anomalies The first stage of capillary network the *plexiform* stage if arrested in its development will produce the capillary angiomas or vascular nevi They remain harmless birthmarks until a sudden connection with the systemic circulation starts feeding them with blood, in which case progressive cavernous dilatations develop They may grow so destructively that bone is invaded and necrotized They are malignant in the clinical sense but do not have the histologic characteristics of a true angiosarcoma.

The second stage of Woollard is characterized by enlarged tubes showing island formation coalescence and tendency to fuse This is the *retiform* stage Capillaries from which the blood has been diverted, atrophy The cavernous

angiomas and the diffuse phlebarteriectasias may derive from this stage of development. Histologically these are numerous parallel vascular tubes, which have not fused sufficiently and are connected by multiple communications. These communications may be of microscopic size or hardly visible to the eye, although rarely they are large enough to produce a palpable and audible thrill. This, of course, is a self-perpetuating and self-enlarging mechanism although, as will be shortly pointed out, certain accelerating factors are clinically recognizable.

In the third stage of definite *stem formation* a persistent anomalous trunk appears, having more or less extensive connections with the systemic circulation. One sees clinically such large anomalous tubes, neither artery nor vein, taking an atypical course in the affected extremity. Maximow made the interesting observation that such a primitive vessel contains bundles of longitudinal muscle fibers just below the intima.[5]

Obviously, transitions from one stage to another do occur, but it is helpful to keep such a triphasic development in mind, since it creates some order in the confusing nomenclature of birthmarks, nevi, congenital arteriovenous fistulae and simple cavernous and racemose angiomata.

Accelerating Factors in Development

There are some readily recognizable accelerating factors in the growth and invasive spread of these vascular anomalies. One is the effect of puberty and pregnancy. Twice we have observed in pregnant women the rapid spread of lesions which were either well controlled or clinically latent. In one boy, whose congenital vascular anomaly was under good control by multiple stage operations, the onset of puberty had an accelerating effect. It is known that estrogen induces a number of vascular reactions reminiscent of those seen in the vessels of the uterine mucosa; the work of S. W. Reynolds has been quoted in chapter 2, Vascular Shunts (p. 9), particularly in regard to coiled and parallel vessels as a pressure-reducing device. One is also reminded of the interesting work of William Bean[6] on the effect of estrogenic substances on vascular spiders occurring in cirrhosis of the liver. At any event, the sexual cycle has a definite influence on the peripheral vascular tree and this may have to be considered in evaluating not only a *mechanical* but an *endocrine* effect on the size and spread of vascular anomalies.

In the section on varicose veins (p. 326) mention will also be made of the spread of painful and tense cutaneous venous spiders which appear early, sometimes as the first reminder of pregnancy, and which are kept under fair check with estrogen therapy.[7] The mechanism of these small angiectasias is unclear; they may represent the opening of small arteriovenous shunts, although they do not pulsate and are distinctly cyanotic.

Of some medico-legal importance is the influence of another factor, namely that of *trauma*. Unquestionably trauma may accelerate a quiescent, inactive birthmark or congenital arteriovenous communication. In a 58 year old man, cited previously,[8] we obtained the history of the patient having

fallen off a hayloft at the age of 12, after which his arm began to enlarge Nevertheless the surgical excision of the ulnar artery with multiple fistulous connections to the veins left no doubt as to the congenital origin of the arteriovenous communication Trauma may also adversely affect the multiple capillary aneurysms which appear as small dark spots in the skin of the port wine nevus These miliary aneurysms bleed profusely at the slightest provocation and may cause acute and chronic blood loss

Pathology

Reid[1] was first to suggest that the so-called angiomata are not neoplasms but in reality are forms of arteriovenous communications Histologic

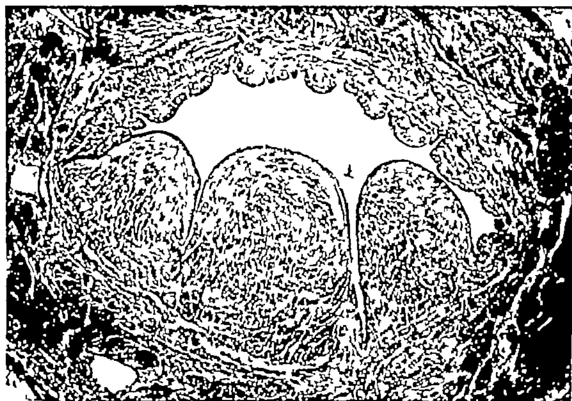


FIG 77 Venous segment from A P., a 21 year old girl, suffering from a congenital vascular anomaly of the lower extremity There is no evidence of endothelial proliferation, but the intima is thrown into large folds as a result of shrinkage after distention, and there is muscular hypertrophy Both of these findings are simply the response to venous hypertension.

sections of these angiomata usually reveal nothing but dilated channels generally veins and the pathologist cannot commit himself as to their origin and nature (fig. 77) It is true however that a few cases of angiosarcoma (hemangioendothelioma) are on record originating from a vascular anomaly These will be discussed in chapter 12, Tumors of the Vascular System

Physiologic Changes

The physiologic changes resulting from abnormal arteriovenous communications differ somewhat from those observed as a result of acquired



FIG 78 Benign capillary hemangioma (Courtesy of Dr Paul W Greeley)

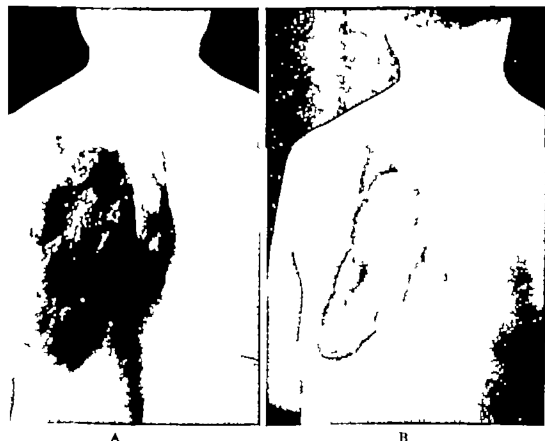
A B

FIG 79 A and B Huge cavernous hemangioma seen at the age of 36 in S B The external carotid artery was divided distal to the superior thyroid artery Multiple injections of sclerosing solution were given into vascular dilatations in the lids, cheek and upper lip Deep x-ray therapy was administered externally and intraorally A wedge-shaped resection of the upper lip and cheek were done The eyelid was later under plastic surgical care (Dr P W Greeley) Patient was re-examined 20 years after surgery His condition was originally described by the county supervisor as horrible, revolting and repulsive "It is hard to understand how Divine Providence could allow such an affliction to befall a human being" Following the operation he married, and now has children and a good job in a Michigan community.

munications Since the congenital fistulae are small often microscopic e effects will be minimal if they exist at all While the hemodynamic con ences in traumatic arteriovenous fistulae will be discussed in detail in ter 8 Arterial Injuries it is worth remembering that an occasional con tal vascular anomaly containing larger than ordinary communications exhibit the bruit maximal at the site of a fistula together with a dilata of the proximal arterial segment the cardiac enlargement and the in se in blood volume In the case of Israel quoted by Emile Holman [9] a congenital vascular anomaly of the right lower extremity was accom ed by a notable cardiac enlargement which receded to normal after the vas amputated by Langenbeck In our total experience with congenital ular anomalies I can only remember two cases in which a palpable and ble bruit was noticed and in none was there cardiac dilatation

cal Picture

The picture varies anywhere from cutaneous nevi (fig 78) to a huge d pulsating deformity This may occupy the entire face in the form of vernous hemangioma (fig 79) the scapular region (figs 80 and 81) the r and lower extremities or the viscera including the brain (fig 82) and ungs (fig 83)



A B

FIG. 80. A and B. Scapular angioma in the case of E.Ch The lesion was first injected with er cent alcohol and followed by complete excision, including some of the eroded ribs a split thickness skin graft was used to cover the defect



FIG 81 The vascular mass has been injected with 35 per cent Diodrast It is hard to be sure of pleural extension, and the mass seems to lie external to the ribs

Typical of the congenital vascular anomalies are the following characteristics (1) they are present at birth or possibly noticed in the first few years of life (2) They exhibit sudden growth, as pointed out before, on trauma, at puberty or during pregnancy (3) The skin usually manifests some port-wine stains, although these are not always present (4) Extremities grow faster and present the problem of arresting their growths at an appropriate time (5) Blood removed from a distended vein is redder (arterialized) than venous blood removed from an uninvolved area This can be readily demonstrated on a blotting paper (6) Venous oxygen saturation is abnormally high as compared to a sample from an uninvolved area (7) An arteriogram or phlebogram hardly ever visualizes the communications since they are very small and multiple However, the site of maximal venous dilatation usually denotes a larger communication

Thus, vascular anomalies may occur in any region of the body The surgeon is more apt to encounter them on the fingers (fig 84), the forearm (fig 85), or the entire upper extremity (fig 86) Our own experience includes find-



FIG 82. Congenital aneurysm of the internal carotid artery in K.W., age 48 who had symptoms since the age of 9 (Courtesy of Dr Oscar Sugar U of Illinois College of Medicine)

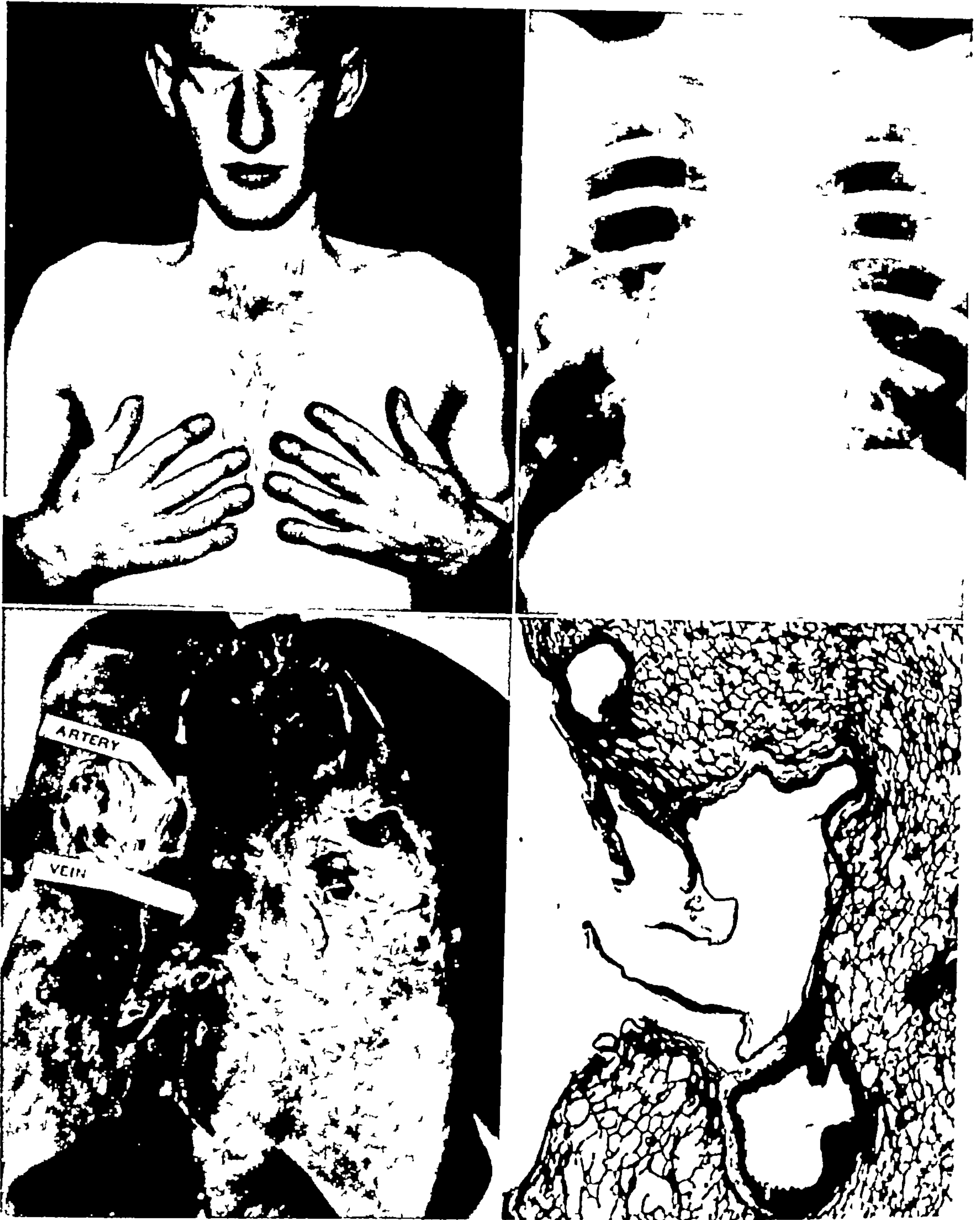


FIG 83 Cyanosis, clubbing of the fingers, large irregular opacity in the left lung field and small opacity near the chest wall in the right lung field In the gross specimen, note the large vascular mass with arterial and venous communications The lower right field shows a microscopic section of a portion of fistula (Courtesy of Dr Wm E Adams J Thoracic Surg, *23* 188, 1952)

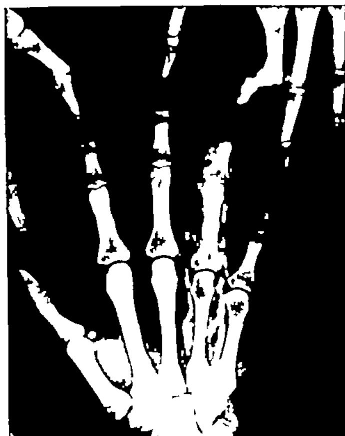


FIG 84 Isolated congenital arteriovenous communications in a 45 year old department store clerk She had recurrent attacks of phlebitis in this finger which repeatedly ulcerated and bled. Three operations were necessary to bring the process to a standstill The visualized vessels are veins with two phleboliths on the ulnar side.

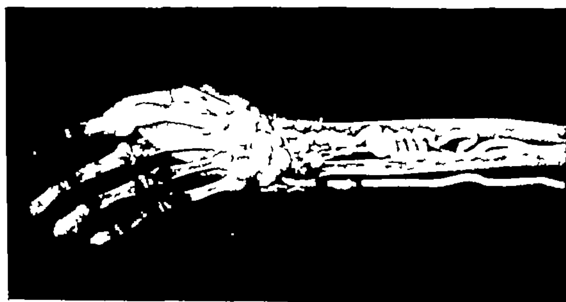


FIG 85 A rather characteristic arteriogram of a localized vascular anomaly of the fore arm. The communications are usually found at the side of maximal elongation and tortuosity the opaque substance is mostly in venous channels



FIG 86 Extensive congenital vascular anomaly of the entire right upper extremity with visible venous dilatations in the subclavicular area There were bruits in this arm and the heart was enlarged Note the huge vascular mass under the clavicle Operation was refused



FIG 87 Note the large left lower extremity with a port-wine stain faintly visible to mid-thigh, huge dilated vessels around the lateral side of the patella, discolored and spongy vascular masses on the toes This limb was 1½ inches longer than its fellow The patient had to wear a larger size shoe on the left



FIG. 88 A huge muscular vessel which ran up to the pelvis in a 28 year old law student. This vessel was stripped in 1945 and three more operations have been done since the leg is useful and painless. Note a large thrombus at ankle level. The exact communications in this case, as in others, can never be positively ascertained at operation. Sometimes a typical artery suddenly loses its muscle and continues as a vein.



FIG 89 An extensive gluteal vascular anomaly with numerous phleboliths The anomaly extended into the pelvis and was treated with roentgen therapy elsewhere. This precipitated menopausal symptoms. Hysterectomy was done, and the mass then shrunk.

ing a large number on the lower extremities (fig 87), where they are frequently associated with a hemihypertrophy of the affected limb, together with cutaneous nevi showing roughly the distribution of dermatomes [9] Such an extremity is longer and warmer than its fellow and shows huge vascular dilatations which, when injected, demonstrate the presence of wide vascular trunks (fig 88) Such a finding, as will be pointed out, directs therapy

Large vascular anomalies of the lower extremities often extend into the buttocks (fig 89), and to the pelvis, and may cause rectal or visceral bleeding and require careful preoperative study so as to anticipate the site and extent of the anomalies Many of them are frankly inoperable

Complications

Bleeding from miliary aneurysms or varices is frequent *Thrombosis* occurs in some of the dilated veins and is exceedingly painful and *intractable*



FIG 90



FIG 91

FIG 90 Huge congenital vascular anomaly with lymphedema and recurrent attacks of lymphangitis Note the differences in leg lengths Multiple Kondoleon operations with excision of large vascular masses resulted in only fair cosmetic but satisfactory functional improvement

FIG 91 This 16 year old girl exhibited an increased length in and a large vascular birthmark over her left lower and upper extremities Note the pelvic tilt The left femoral pulse was absent, the lower leg and foot were cold and shiny, and the skin atrophic A lumbar sympathectomy was done to improve collateral circulation She married and was restudied 10 years later Her only complaint was that of backache A lengthening operation on the right was considered but not accepted

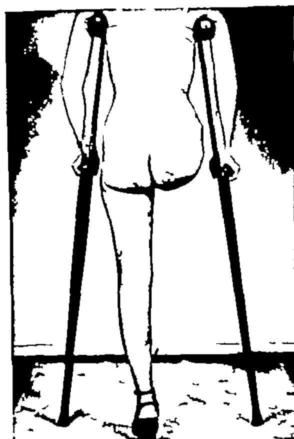


FIG 92. This 20 year old girl, after having been subjected to multiple meddlesome and unnecessary operations, was rehabilitated by a hemipelvectomy (Drs. Chandler and Heck of the Orthopedic Department of the University of Illinois College of Medicine) She married, delivered a normal child with ease and gets around well on crutches.

The affected extremity shows increased growth and together with the venous edema may be the site of recurrent attacks of lymphangitis thus leading to an elephantiasis (fig. 90) This may happen all the more frequently since lym phatic anomalies often accompany the vascular ones so that a *lymphatico-vascular anomaly* is present

Once in a while such an elongated extremity afflicted with a birthmark loses its femoral pulse so that it becomes cold, atrophic and ischemic (fig 91) It then must be treated as any other chronic arterial occlusion

If untreated the anomaly may make a social outcast of the patient (fig. 79) produce a great deal of pain frequent hemorrhage or painful thrombosis and call at times for amputation or even hemipelvectomy (fig 92)

Treatment

All birthmarks must be promptly treated no matter how small Failure to treat them early may render them later inoperable Radiation, radium packs and radium seeds are widely employed by dermatologists and pedia tricians Only the widespread cutaneous port wine stains should be so treated and some fading can be accomplished by carbon dioxide snow or roentgen ray therapy

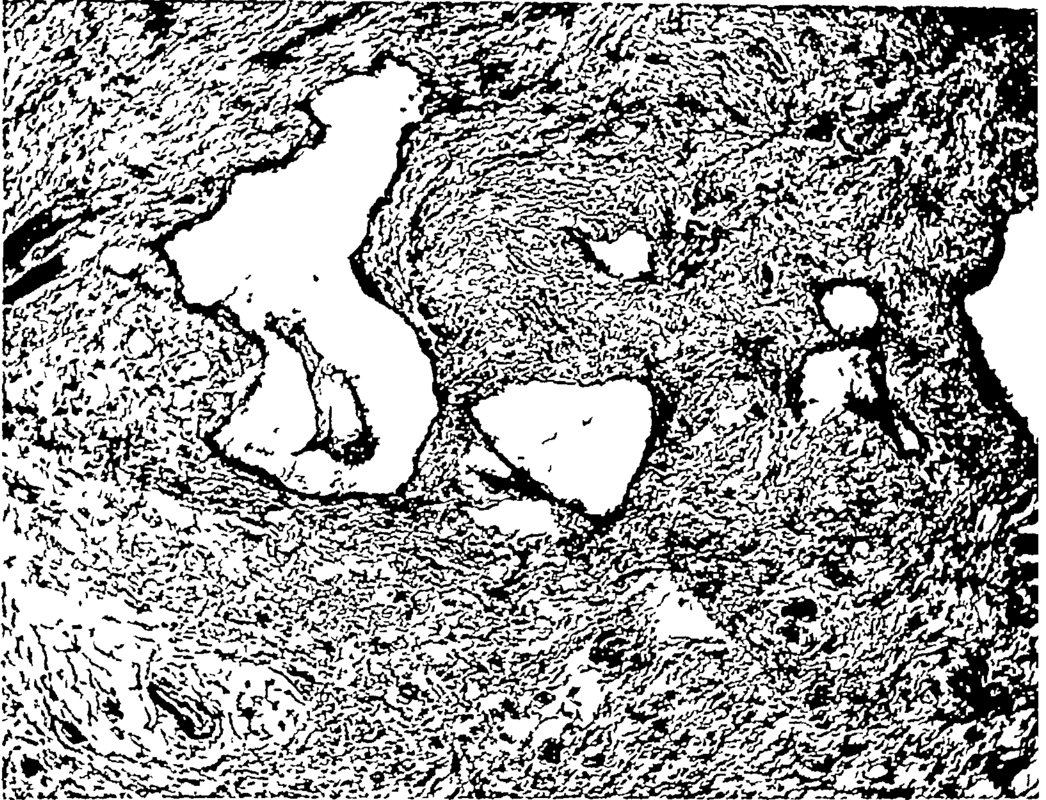


FIG 93 Photomicrogram of tissue excised from the lip of the patient shown in fi
Noted the marked fibrosis produced by preliminary sclerosing injections Excision was greatly facilitated

The localized "angiomata" should be excised and if necessary
graft must cover the resulting defect If the lesion is on the face a prelir
ligation of the external carotid artery or that of the external maxillary
is very helpful One can also make use of preliminary sclerosing inje
with sodium morrhuate, or more recently with 3 per cent Sotradecol
the sclerosed vessels with surrounding fibrosis can be more easily e
(fig 93) When huge areas are excised, the resulting defect must be cc
by a split-thickness skin graft, preferably immediately, although the c
base may force postponement until a dry field is obtained In the extrer
operations should be started as soon as the child can tolerate anesthes
blood loss, preferably after the first year The parents must understan
multiple stage operations are being contemplated in order to arrest the
ess and that amputation is sometimes the best solution, eliminating fre
hemorrhages, much suffering, prolonged disability and mounting exp

After a preliminary study of the cardiovascular system, an arteric
or aortogram is performed Most help is obtained here from localized c
tions of the venous segment (fig 94) Often the major arterial pathways
entirely normal, the lesion being connected with an unabsorbed prir
pathway Demonstration of the arteriovenous communications inva
fails If the phlebogram shows a large single trunk, as it so often does,
stripped from ankle to the root of the limb It is important, however, i
do too much at one sitting and to continue operations once or twice a
combined with sclerosing injections or radium therapy for the cutan

bleeding aneurysms With such a method I have seen a child first seen at the age of 7 go through puberty marry and have a baby by cesarean section She is now 42 years old and gets around well wearing an elastic hose Similarly one county judge and one corporation lawyer who both hunt and play golf expertly have been subjected to multiple operations and may still require further surgery On the other hand a number of upper and lower extremities have to be amputated especially if the main artery feeding the anomaly had been ligated Emile Holman[9] pointed to the disastrous effect of proximal arterial ligation in any kind of arteriovenous fistula, congenital or acquired Figure 95 illustrates the result of diverting the collateral arterial flow through the communication back toward the heart so that the terminal vascular bed will receive no blood We have observed two extremities admitted to our service with frank gangrene after the proximal arteries have been ligated When proximal vein ligation is added this might slightly aid the maintenance of some pressure in the terminal circulation but is certainly not a recommended procedure The problem of concomitant vein ligation in case of arterial ligature or thrombosis will be dealt with in chapter 8 Arterial Injuries

The problem of when to inhibit the growth of a more rapidly growing extremity affected by a vascular anomaly has been greatly facilitated by the studies of Dallas Phemister[10] While ½ to ¾ of an inch of added length



FIG 94 The first stage of the operative procedures on S.K. a 19 year old girl, consisted in the excision of a diffuse vascular mass below the lateral malleolus. Higher segments were stripped.

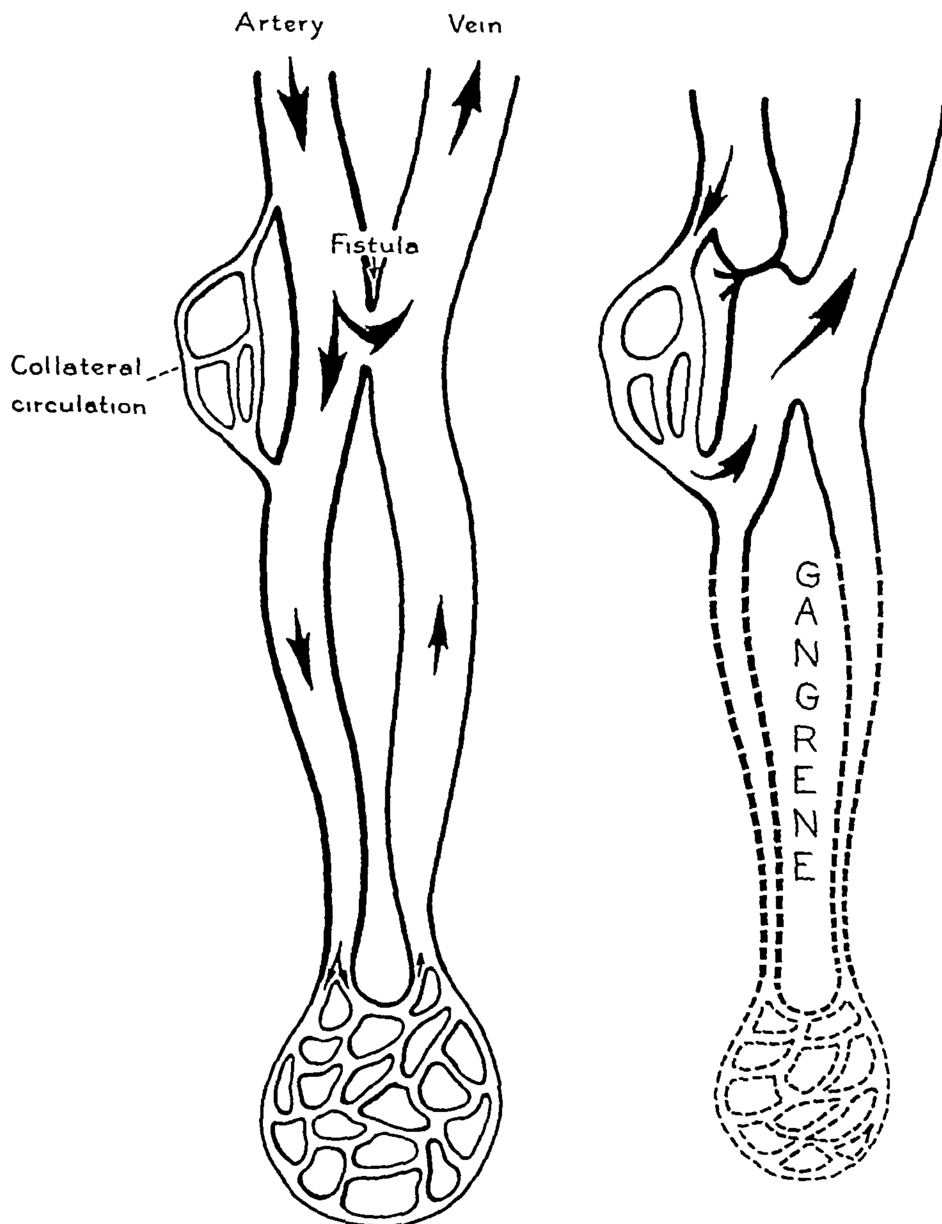


FIG 95 Diagram of the effect of proximal arterial ligation in the case of an arteriovenous fistula On the left, much of the arterial blood still flows to the capillary bed, both through the main and through collateral pathways On the right, the main artery has been tied proximally to the arteriovenous fistula The collateral blood supply with its diminished flow and pressure will flow into the vein offering least resistance Peripheral flow into capillaries is minimal and gangrene results (Modified from E Holman Arteriovenous Aneurysm The Macmillan Co, New York, 1937)

can be compensated by adding a heel to the opposite, normal extremity, such congenital anomalies may produce a difference of 2 to 3 inches in length with all the untoward effects of a pelvic tilt. For this reason, in several children under our care, the orthopedic service carried out an epiphyseal arrest at a time when the affected limb had acquired a length that the unaffected limb was expected to obtain in years to come.

This operation is simple and can be readily sandwiched in between the multiple resections of the arteriovenous shunt Its proper timing takes mature orthopedic judgment [11]

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CHAPTER 8

ARTERIAL INJURIES

ARTERIAL INJURIES, IN THE BROAD SENSE OF THE WORD, WILL BE DISCUSSED IN this chapter Following those due to mechanical trauma, physiochemical arterial injuries due to heat, cold, chemicals, electric currents and radiation will be dealt with

1. MECHANICAL INJURIES

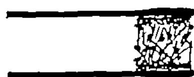
Arterial injuries have always occurred in comparatively large numbers during warfare Running out of hot oil, Ambroise Paré first used his arterial ligatures as a military surgeon Following World War II, six university centers conducted a follow-up study of 802 arterial injuries sustained by 741 men [1] Much was learned then, but the more recent Korean conflict supplied material for the evaluation of direct restoration of continuity [2] Never before, however, has there been such a tremendous increase in civilian injuries due to automobile traffic, hunting accidents and violence, so that the lessons of the wars must be adapted and utilized in dealing with the traumatic surgery of civilians The quality of adequate systematic treatment of these injuries by some civilian hospitals is still not up to the standards developed during warfare, nor can these standards always apply to civilian injuries The military surgeon, used to a young, sturdy group of injured who come to operation within a period of hours or days, is greatly disappointed when his experience needs modification in civilian practice The patients are older and their delay in obtaining definitive care is obvious, although much progress has been made, mostly through the efforts of the Committee on Trauma of the American College of Surgeons

CLASSIFICATION

An artery may be crushed, severed, lacerated or contused [1] The response to an injury of a vessel is contraction which closes, at least temporarily, the defect in the vessel wall The blood clots because all three of the clotting factors, *i e*, injury to the intima, slowing of circulation and injury of surrounding soft parts liberating thromboplastic substances, are at work The platelets deposited on the lining of the injured vessel liberate a potent vasoconstrictor, "serotonin," which adds to constriction of the vessel and may be


a. Platelet agglutination


b. Appearance of fibrin


c. Fibrin clot


d Clot retraction

FIG 96. Hemostasis in a severed vessel (a) platelets agglutinate at a raw endothelial surface (b) fibrin appears sealing the cut end of the vessel (c) fibrin clot and vasospasm effect a firmer closure (d) clot retracts and serotonin approximates the cut end. (Modified from Quick, A. The Hemorrhagic Diseases and the Physiology of Hemostasis. Charles C Thomas, Springfield, Ill. 1942.)

a very important factor in hemostasis (fig 96) When the vessel is completely severed the two ends retract owing to the longitudinal tension in the artery The phenomenon is seen regularly on transecting arteries for the purpose of arteriectomy or resection and replacement It is generally agreed that a partially split artery bleeds more readily than a completely severed one In large arteries this natural hemostatic mechanism fails and bleeding ensues into the surrounding tissues A contused artery develops an intramural hematoma which may dissect under the adventitia or should the muscle be partly torn under the media. It may also go into spasm and later produce stenosis or thrombosis, especially if the artery is affected by an inflammatory or degenerative arteriosclerotic process This aggravation of a pre-existing vascular disease by trauma is not uncommon

A massive arterial thrombosis following a comparatively mild injury always calls for a thorough examination of the patient s vascular status not only that of the vessel wall but of the blood itself Thus, polycythemia ex hibits peripheral arterial thrombi in many regions and a minor injury may contribute to their localization [3]

Clinical Picture

An arterial injury may be *obvious* when an area of a major pathway is pierced by a bullet, shattered by a grenade crushed by a collapsing building or stabbed by a revengeful suitor all of these are followed by massive bleed ing to the outside, by the classic signs of acute blood loss or by a growing hematoma exerting pressure on surrounding structures Sudden loss of blood is usually compensated up to 1000 cc by vasoconstriction and speeding up of the heart rate Only when this line of defense fails will blood pressure

begin to fall As it reaches a "critical" level of 80 mm of mercury, hunger, thirst, cold clammy extremities, disappearing pulse and finally loss of consciousness develop Hemorrhagic shock now reaches an irreversible phase, for even if blood volume is rapidly replaced, widespread capillary damage is present, so that plasma will ooze out of the vascular system as fast as blood is poured in. Also the function of important organs, such as the brain, heart, kidney and liver, are so damaged by the hypotension that recovery can only be partial or incomplete

It is important to realize that *fall in blood pressure* and even *tachycardia* only develop after an elaborate compensatory mechanism of the body has been overcome; in fact bradycardia may be seen in hemorrhagic shock with fall in blood pressure

In addition to the signs and symptoms of actual loss of circulating blood volume, reflex effects from the injured area, from pain, anxiety and exposure to cold produce *neurogenic shock* Widespread capillary damage occurring in the "crush syndrome" produces protein loss and hemoconcentration, just as in a burned area Severe vomiting and sweating still further decrease blood volume

Since blood volume determinations, even if available, only reveal a large portion of the true blood loss,[4] the appearance of the patient is most important

The Clinical Research Unit of the Medical Research Council of Great Britain, under the direction of R T Grant, has described a number of circulatory patterns which characterize the clinical picture of patients suffering from loss of blood [5] The four signs used were (1) the level of systolic blood pressure, (2) the pulse rate, (3) the temperature of the extremities, and (4) color of the face

The normal circulatory pattern was defined as a normal blood pressure (100 to 140 mm Hg), a normal pulse rate (70 to 99 per minute), warm extremities and a good face color In injured patients such a picture is associated with little blood loss and a blood volume nearly normal In patients with larger blood loss, this pattern should develop after adequate treatment Grant and Reeve, on the basis of close clinical and laboratory study of 230 patients seen during the London air raids in 1940 and during the Italian campaign from 1944 to 1945, have established six abnormal patterns While these are obviously somewhat diagrammatic they are of great aid in immediately sizing up the clinical situation, particularly if no laboratory aid is available The main disturbances of the normal pattern are six In two the blood pressure is normal, in one it is raised and in the remainder it is low The following description of these patterns is quoted verbatim.

(1) "The pattern of *cold tachycardia* is a normal blood pressure, a fast pulse rate, cold extremities and usually a pale face It is met with chiefly in the first few hours after injury and is then commonly associated with a moderate blood loss and blood volume reduced to between 70 to 80 per cent of normal It is also the pattern associated with the constrictor phase of a transfusion reaction

(2) The pattern of *warm tachycardia* is a normal blood pressure, a fast and bounding pulse warm extremities and usually a well colored but some times a pale face It is associated with a blood volume of 70 per cent normal or over and is met with (i) in the dilator phase of a transfusion react on and (ii) in injured patients in whom hemoglobin has fallen to very low levels

(3) *The hypertensive pattern* is a raised blood pressure and usually a normal or slow pulse rate The extremities may be warm or cold and the face well colored or pale It is associated usually with a small blood loss and a blood volume of 80 per cent of normal or more Usually transient, it is often met with soon after injury but may also occur before and during operation if suitable stimuli are applied

(4) *The vasovagal pattern* is a low blood pressure a slow pulse rate cold extremities and a pale face To these may be added sighing respirations sweating, nausea and vomiting. It is commonly met with soon after injury is transient and is due to sensory and emotional stimuli rather than to blood loss Less commonly it occurs both before and during operation when suit able stimuli are applied Occasionally it is associated with much blood loss and may be seen as a terminal pattern in patients dying from hemorrhage

(5) The pattern of *cold hypotension* is low blood pressure, a fast pulse rate, cold extremities and a pale face It is found in two types of cases (i) It is commonly seen in patients whose blood volumes are reduced by hemor rhage below 70 per cent normal and then tends to persist until abolished by transfusion In patients with great blood loss and a blood volume reduced below 60 per cent of normal an extreme form develops namely a very low blood pressure (under 70 mm Hg) impalpable pulses and a very rapid heart rate, cold extremities with constricted veins and pale face and lips To these may be added great restlessness dyspnea and sweating. This form indicates the need for immediate large and rapid transfusions to save life (ii) It is met with in patients suffering from heavy infection such as advanced peritonitis and in them may be associated with a normal blood volume and is not abol ished by transfusion Here the pulse rate tends to be faster and the extremi ties warmer than when hemorrhage is the provoking factor

(6) The pattern of *warm hypotension* is low blood pressure a fast pulse rate and warm extremities The face may be flushed well colored or pale This state is usually but not always transient Blood volume is usually reduced, although not below 70 per cent normal The factors provoking it are not properly understood It is usually met with in warm surroundings often after operation, when it is thought to be due to a combination of factors such as anesthetic agent (ether and cyclopropane) body warming and previ ous transfusion It is not uncommon during operation, when ether and cyclo-propane are probably concerned and is sometimes brought about by undue body warming before operation

These patterns have been described in full because of the conviction that in most civilian injuries the extent of hemorrhage following limb or abdomi nal injuries is underestimated That hemoglobin and hematocrit determina tions are useful need hardly be stressed but serial blood volume determina

tions, especially during military activity or mass disasters, will seldom be feasible

Patterns (3) and (4), the hypertensive and the vasovagal, are obviously of neurogenic origin One can only see them shortly after injury or by accidentally witnessing massive hemorrhage in the hospital.*

During operations, which are forms of vascular injury, these neurogenic reactions are mostly suppressed, thus lulling the surgeon and anesthesiologist into a false state of safety Only too often does one hear the statement that the patient's hands are warm, he is all right, when with a rising pulse and a falling blood pressure he is in a state of *warm hypotension*

Not all arterial injuries, however, produce such obvious clinical pictures Masked arterial injury occurs in patients in whom the pressure of the extravasated blood can counteract, temporarily at least, further bleeding (pulsating hematoma), in whom the artery and vein are simultaneously perforated so that an arteriovenous fistula develops, or in whom the blood clots and seals not only the tear in the artery but the whole lumen, and results in *traumatic thrombosis* which is fairly well compensated by collateral circulation

The *contused or spastic artery*, developing in the vicinity of a soft tissue or bone injury, often leads to final thrombosis (p 143).

Associated Injuries

Arterial injury is often complicated by nerve injury, fractures, a massive tear and destruction of muscles On the upper extremity, nerve injury is present in roughly 75 per cent of the cases, but on the lower extremity only 40 per cent of arterial injuries have nerve involvement [1] Multiple arterial injuries in the same limb are present in about 10 per cent of the cases and this is sometimes overlooked in their revision Fractures and infections of massive soft tissue injuries, of course, worsen the prognosis for the limb Fractures themselves can cause arterial injury, as can dislocations, particularly around the elbow and knee Arterial injury may also occur as a result of a tourniquet (fig 97) or tight cast, although, of course, nerve interruption is more frequent and more widely feared

In civilian injuries, the median age of the population is much higher than that of the men wounded in service (*i e*, 23 to 27 years) Blunt injuries in the older patient, or percussion waves from a bullet not actually piercing the artery, are more apt to create thrombosis and there is no question that the arteriosclerotic artery is more vulnerable Even a careful dissection of an artery or traction with cord tape may fracture a plaque or produce a subadventitial hematoma Artery clamps below the aorta frequently produce vessel damage A minor injury to a toe may be followed by gangrene when

* I will never forget a row of severely injured Austro-Hungarian cavalry men in blue tunics and red breeches lying on stretchers in a cold churchyard following an attack on heavy Russian artillery This was in the fall of 1914 with no transfusions and no antibiotics, only subcutaneous physiologic saline drip The ones with cold hands, thready pulse and air hunger were not taken to the operating room but given morphine and allowed to die in peace Their condition was regarded as irreversible

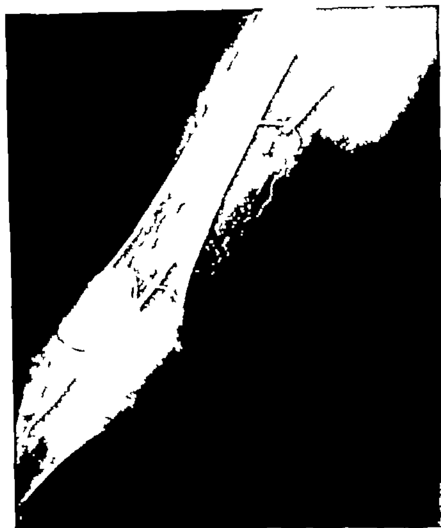


FIG 97 Arterial interruption in a 54 year old woman who showed tourniquet paralysis after a bunion operation. She had some peripheral arteriosclerosis but developed a pulseless foot immediately after an orthopedic procedure, done under tourniquet. Note atheromata above and below the occlusion.

the arterial circulation is diminished The increased capillary fragility of the hypertensive and diabetic patient will produce large hematomas interfering with collateral circulation, especially in tight compartments such as the axilla, the popliteal space or between the fibrous septa of digits

Type of Injury

Arterial interruption false aneurysm (pulsating hematoma) arteriovenous aneurysm, arterial contusion and arterial stupor (traumatic vasospasm) are distinct types and need distinct treatment Hence their early recog nition is important They may often be found in combination with each other

Arterial Interruption

This will produce a degree of ischemia which depends on the site of the injury on the extent of soft tissue damage with edema and on the exte

traumatic thrombosis proximal and distal to the injury Needless to say, the maintenance of adequate blood pressure is of primary importance and prolonged hypotension is a serious menace to the viability of a limb or organ whose major artery is interrupted. Generally speaking, acute interruptions on the upper extremity are much better tolerated, develop better collateral circulation and do not ordinarily need restoration of continuity. Certainly the changes in color, the drop in skin temperatures and the loss of pulsations, together with the complaints of numbness and pain, are much more intense in the lower extremity Injury to the axillary artery, however, especially if associated with an axillary hematoma, can so throttle the two important collaterals that critical ischemia or even gangrene may result The dangerous segment here is between the subscapular and circumflex vessels The popliteal artery represents a dangerous segment, for its closure is followed by amputation in 85 per cent of the cases

Pulsating Hematoma

A false aneurysm, or pulsating hematoma, occurs when bleeding takes place through a partial tear or complete severance into a preformed tissue space where blood clots, thus limiting, at least for a while, the expansion of the hematoma The center of the clot retracts, becomes liquid and shows a transmitted pulse together with a systolic bruit An interesting lining forms the wall of the pulsating hematoma, a pseudo-intima, not unlike the one which forms inside plastic or metal prostheses used to bridge arterial defects. A rupture of this false sack occurs, or it may be incised to "drain an abscess cavity," an occurrence witnessed on three occasions

The extravasated blood may first effectively seal the rent in the artery but a slow leak may increase the palpable mass A thrill or bruit, definitely systolic and not to and fro, is heard. The closed spaces at the axilla, groin or popliteal space limit expansion, but will compress venous outflow and produce damage to the nerves Collateral circulation is so seriously hampered that irreversible ischemia develops The damaged main channel may undergo degenerative changes, notably extensive calcification Infection temporarily controlled by antibiotics will thrive in such a culture medium For this reason the diagnosis of a pulsating hematoma calls for urgent intervention, a rule which, unfortunately, is often ignored in civilian injuries

Arteriovenous Aneurysm

A traumatic arteriovenous fistula occurs when a connection is established between an artery and its concomitant vein 30 per cent of 802 arterial injuries sustained in World War II exhibited arteriovenous communications, with 0 4 per cent multiple fistulae, one naturally wonders about the conditions which favor such a complication Obviously, large shattering explosive missiles will seldom produce arteriovenous fistulae, whereas gunshot wounds, stab wounds or fragments of bone are more apt to do so Since

the hematoma may be small and the leaking of blood into the venous system occurs either immediately or within a few days after the injury large masses do not develop In fact, many of these wounds are debrided and the vascular lesion is missed Whether or not a simple arterial injury with a growing pulsating hematoma may later erode the vein and create a secondary fistula is difficult to prove but is a distinct possibility The hemodynamic disturbances following the establishment of an arteriovenous fistula have been intensively studied by Emile Holman [6] and form the basis of clinical signs and symptoms to be looked for

The arterial current will follow the path of less resistance and thus will pour into the vein and only partially into the distal arterial segment (see fig 95) The factors determining the volume flow through the fistula are the *size of the opening its location in the main arterial tree the absence of fibrosis around the fistula* and *the duration of the fistula* A fistula between the aorta and vena cava will cause rapid cardiac failure but the same sized opening between the femoral artery and vein may progress for years without doing so Cardiac dilatation and decompensation are reversible when the fistula is closed (fig. 98)

An extraordinary development of collateral circulation appears in the injured limb The artery proximal to the fistula is dilated the artery distal to the fistula carries blood in a cephalad direction if the size of the fistula is larger than the diameter of the artery just proximal to it When the artery just distal to the fistula is ligated collateral circulation is minimal or fails to develop

When the experimental studies of Holman are transferred to clinical observations, it may be said that in addition to the duration of the fistula, which in our experience may have been as long as 15 to 30 years (fig. 99) the



FIG 98 Two meter chest films of K.M suffering from a traumatic arteriovenous fistula of the tibioperoneal artery and vein of 21 years duration (A) before, and (B) two weeks after quadruple ligation and excision of fistula.



FIG 99

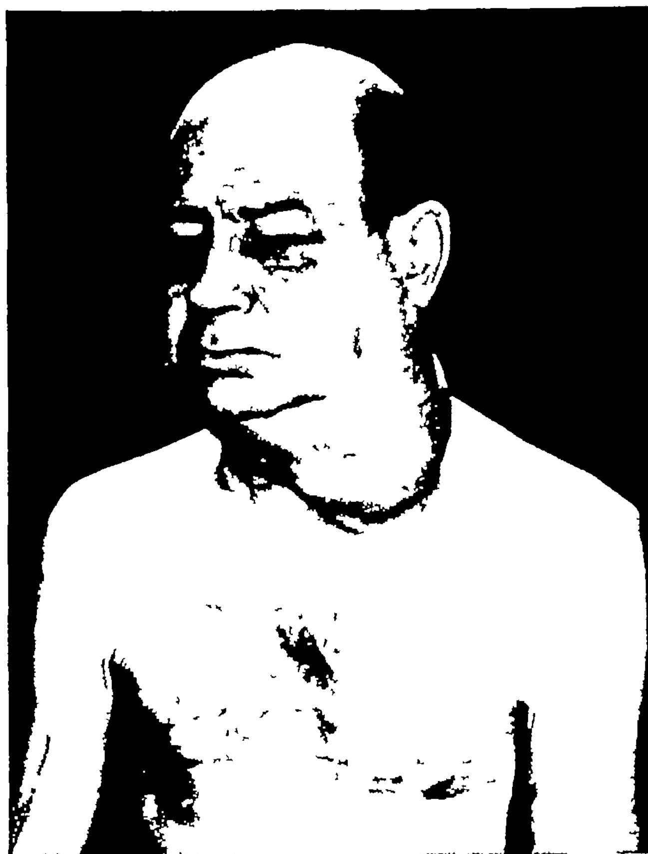


FIG 100

scarring around the fistula is the one factor that may inhibit the development of adequate collateral circulation In the fistulae of long duration which military surgeons do not see there is extensive dilatation arteriosclerosis and even aneurysm formation of the proximal segment which make a direct arterial repair sometimes impossible Even ligations are hazardous and in one case ligation led to a secondary hemorrhage

Distal to the fistula, the injured extremity is swollen warm and full of large varicosities and occasionally ulcerations The tips of digits may be cyanotic and cold since the majority of the blood flow is through the fistula back to the right heart The blood volume is greatly increased (fig. 100)

Arterial Contusion

The injury of the artery may extend proximally and distally to the visible laceration or severance as emphasized by Jahnke and Howard [7] The artery may also suffer from a blunt injury or bullet causing an intramural hematoma Such injuries are far more significant in patients with sclerotic vessels who show a tremendous development of vasa vasorum which may rupture even spontaneously (fig 101) The possibility of aggravating a pre-existing vascular disease by a comparatively minor injury is thus very real Threading polythene tubes into sclerotic femoral arteries for retrograde aortography (p 96) which requires a 14 to 16 bore needle is therefore to be undertaken with very strict indications An arterial puncture with a number 18 needle under direct vision with avoidance of atheromatous plaques is distinctly preferable

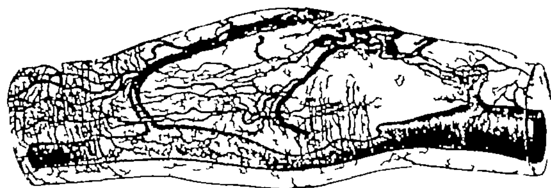


FIG 101 Cleared specimen of stenotic, atheromatous renal artery The vasa vasorum are fed by collaterals and show a marked development. They are vulnerable and often rupture spontaneously (Winternitz, et al. The Biology of Arteriosclerosis. Charles C Thomas, Springfield, Ill., 1938)

FIG 99 Angiogram of arteriovenous fistula below the knee The sac is well visualized and so is the marked venous engorgement below the communication. (K.M St. Luke's Hospital, Chicago, Oct. 21 1940)

FIG 100 Traumatic arteriovenous aneurysm of the neck in a 62 year old hotel clerk following a gun-shot wound of 30 years duration. Note the venous distention on the anterior wall of the chest and upper arm. The apex of the heart was in the midaxillary line He was decompensated. An operation was refused.

Such arterial contusions may be subclinical, but may also be followed by occlusive thrombosis. Myogenic spasm, to be discussed presently, may be the precipitating factor. Acute arterial contusions followed by thromboses have been described in great detail by Leriche.[8]

Arterial Stupor

For many years the neurogenic vasoconstriction of the traumatized or embolized artery was thought to be predominant.[8] It has become apparent, mostly through the writings of Cohen[9] and Kinmonth,[10] that in addition to vasomotor reflexes a myogenic contracture of the injured vessel occurs, especially around fractures or dislocations near the elbow or knee and under other conditions which do not directly injure the artery itself. While only infrequent observations can be made immediately after injuries, the recent opportunities to explore and handle arteries and veins for purposes of restoring continuity have given adequate material for observation. In some patients, merely touching the wall of the vessel with a blunt dissector may bring on a visible spasm extending over several centimeters in length; this will readily occur in a sympathectomized extremity.

Diffuse aching pain, followed by pallor, coldness and loss of pulsation develops, all typical of a sudden arterial occlusion. If this is a reflex type of arterial spasm a paravertebral block will abolish it. Papaverine may be given intra-arterially or intravenously in ½ gr. doses to abolish the suspected spasm.[11] Should no release of arterial flow occur, only a direct exposure can verify the presence of a myogenic spasm as against a thrombotic occlusion.

A combination of segmental arterial spasm, contusion and late thrombosis with infarction of muscles surrounded by tight fascia characterizes Volkmann's ischemic contracture. Griffith's Hunterian lecture on this mysterious malady, delivered in 1940, deserves the careful study of everyone interested in this subject.[12] He believed that more important than the loss of the main channel is the spasm of the collateral circulation. Thus it is necessary to de-emphasize extrinsic pressure, splints or hematoma formation, and focus attention on the release of vasospasm in the main and collateral channels.

TREATMENT

Some of the old concepts governing the surgical care of vascular injuries have undergone radical revision. In general, vascular injuries were treated by delayed surgical management, waiting for collateral circulation to develop. This principle was certainly justified at the time when ligation of the injured artery and quadruple ligation and excision of the arteriovenous fistula were practiced; this was true to a great extent during and immediately following World War II. DeBakey and Simeone have given an extensive account of the results obtained and Elkin and DeBakey have edited a most enlightening monograph, *Vascular Surgery in World War II*, as part of the series published by the Medical Department of the United States Army.[13]

Daniel C Elkin[14] has reported on 340 arteriovenous fistulae of these 338 had been treated with quadruple ligation and excision and 12 cases were repaired While there was not a single amputation in this series, vascular insufficiency remained in 30 per cent of the cases In civilian injuries of many years standing, this procedure may still be the method of choice when the dilated paper thin artery is unfit for anastomosis and extends proximally for a great length interfering with resection and replacement

There were however a number of successful reconstructions of arterial injuries in army and veterans hospitals after the end of World War II and Freeman and Shumacker were the early champions of this trend [15] Important details involved in the immediate care of arterial injuries emerged from the Korean War [7]

Upon arrival to a hospital equipped for arterial repair (a station hospital in the theatre of operations a hospital ship or a general hospital in civilian practice) the patient is immediately given a transfusion if with nothing better with type O blood until pulse and blood pressure are stabilized and the normal circulatory pattern is established [5] It is almost best to do this in the operating room of a civilian hospital while the often cumbersome apparatus of a general hospital is set in motion for an emergency procedure The tourniquet, if present is removed and direct pressure with a large bandage is instituted A portable roentgen ray picture is taken to ascertain the presence of fracture or retained foreign bodies Blood must be given preferably through three or four bottles running at once General anesthesia must be induced slowly without a stage of excitation with intravenous Pentothal sodium This drug may induce a fall in blood pressure If control with the tourniquet is impossible proximal arterial control should be established by a separate incision well above the site of injury using cord tape or rat tooth clamps for temporary interruption The injured vessel must be widely exposed establishing proximal and distal control with bulldog clamps Whenever possible, arterial continuity is restored but if laceration is extensive it is far better to resect the injured vessel and reanastomose it when this can be done without tension Rather than suture the artery under tension, a vein graft from the patient or a frozen homologous artery* must be used to restore continuity This is true for pulsating hematomata and for arteriovenous fistulae except that in the latter case no attempt need be made to restore venous continuity The veins are ligated, proximally and distally to the fistula. Transvenous suture in late civilian injuries has been used by us a number of times In case of spasm of the artery it is wrapped in a sponge soaked in 2 5 per cent solution of papaverine according to the method of Kinmonth.[10] The reconstructed artery should be surrounded with soft tissue but aside from that the soft parts and skin are left wide open for secondary closure 5 to 10 days later This precaution is not necessary if surgical repair is done weeks or months after the injury There is every reason however to urge immediate debridement and repair of all injuries in which arterial injury is manifest or suspected

* See vascular prostheses in chapter 20

Postoperatively, full antibiotic therapy is instituted The lower extremity is immobilized in a posterior molded splint, the upper extremity is bandaged to the chest wall If the color of the extremity is poor on completion of the operation, intermittent or continuous sympathetic block is instituted If the extremity still looks ischemic, re-exploration must be done Should the vessel be thrombosed at the anastomosis, it has to be reopened or a bypass constructed with end to side anastomosis proximal and distal to the thrombosed segment.

With this type of management, the amputation rate fell from 62 per cent following ligation[13] to 7 per cent following repair [7] All amputations following repair occurred in the lower extremities Failure is dependent on technical errors but also on muscle necrosis which occurs prior to operation An anastomosis may be patent and the pulses distal to it full and bounding, yet gangrene of the muscles may require amputation This finding indicates the urgency of arterial repair within six to eight hours, if possible

Breakdown of the arterial repair is usually caused by infection Associated injuries to nerves and bone may have to be ignored in exsanguinated patients, and be repaired later In deliberate, elective procedures the open or closed reduction of bones and the suture of several nerves may be done in one sitting

This Korean experience, as urged by Emile Holman,[6e] should be applied to injuries in civilian practice All penetrating wounds suspected of involving the major arteries to a limb ought to be explored immediately, with the purpose of repairing or reconstructing the damaged artery While a rapidly expanding pulsating hematoma with increasing pain and signs of nerve compression obviously calls for immediate surgical care, many of the arterial wounds are unsuspected and ignored, and only a critical ischemia, appearance of a thrill or a growing hematoma will bring them to the surgeon In my own experience, a stab wound of the superficial femoral artery was treated with weeks of compression, and later the pulsating hematoma was incised for drainage of an abscess, in another case a gunshot wound of the axilla, causing an immediate large hematoma and brachial plexus injury, was allowed to go for four months without definitive treatment This pulsating hematoma was also incised, with massive propulsive hemorrhage occurring through the incision In another case a pulsating hematoma of the thigh was left untreated for 12 years, until a pregnancy activated the mass which required excision and successful grafting Many of the patients arrive with limbs packed in ice, or with tourniquets left in place for 10 to 12 hours. Arteriovenous fistulae have been admitted to our service of 8, 12 and 30 years duration with cardiac failure and with proximal arterial segments which defied all attempts at restorative surgery

In the last cases, excision of the arteriovenous fistula with lumbar sympathectomy results in a viable limb, but the percentage of ischemic symptoms, notably those of intermittent claudication in the lower extremity, is around 30 per cent [1]

The treatment of arterial contusion which probably is present around all arterial injuries is best managed by a segmental resection of the artery proximal and distal to the lacerated or severed vessel in other words with adequate debridement of the vessel [7]

Arterial stupor often only diagnosed or excluded by direct exploration is best treated by Kinmonth s method with 2.5 per cent papaverine Procaine or hot saline packs around the spastic artery have often failed in my experience It is possible that anoxia or CO_2 accumulation facilitates this myogenic spasm but direct experiments bearing on this problem are unknown to me It is certain that this myogenic arterial contracture does not occur in most injuries When it does occur it may be most intractable and cases have been recorded in World War I which necessitated amputation without finding any organic obstruction

In cases where arterial insufficiency is associated with bone or muscle injury especially in the tight compartment about the elbow immediate exploration of the brachial artery and also of the median nerve is indicated If the artery is swollen and contused one should not hesitate to resect it as suggested by Leriche [8] In most of the cases of Volkmann s contracture in which the artery was inspected early it was found to be narrowed to a string, this spasm extending into the radial and ulnar branches [16] Arteriectomies have been performed especially by Leriche and his school [8] As pointed out earlier the lesion consists of spasm contusion and final thrombosis, with ischemic contracture of the flexor muscles While angulation of the artery over the proximal fragment and hemorrhage into the cubital fossa interfering with venous return together with rough and unskilled reduction and tight splints are obviously important factors in creating muscle ischemia, the syndrome does develop in the absence of any of these factors The important review of Hill and Brooks must be remembered [17] they found 123 cases of Volkmann s contracture without the previous application of any splint and bandage and 28 cases in which there was neither fracture nor constricting bandage or splint. Orthopedic literature unfortunately does not stress exposure of the brachial artery in the early hours of the syndrome nor are the butcher s injuries of Gage and Ochsner[18] (which result from a slipping of the knife while skinning beef) resulting in a stab wound of the upper medial aspect of the thigh ever explored early enough in my experience The acute contusion and thrombosis of the anterior tibial artery resulting in necrosis in the anterior tibial compartment, have also been recognized as the result of injury [19] It may be difficult to differentiate this from an acute thrombosis in Buerger s disease a mistake made by our service in one instance

The type of management advocated by Edwards and Lyons[20] for injuries above the knee or elbow which are followed by absent or diminished pulses is all too often delayed or neglected If the pulse does not return a few minutes after brachial plexus block or spinal anesthesia, the injured artery is explored The perivascular sheath is open widely and a hematoma, if present is evacuated If local application of warm saline procaine or papaverine does not release the spasm, the contused spastic artery is resected Saphenous

vein graft is used in areas with muscular support, and arterial homografts are used in unsupported areas Bypass procedures might lend themselves to sidetrack contused vessels, but, to my knowledge, such a procedure has not been reported in this particular situation

2. PHYSICOCHEMICAL INJURIES

This section contains the discussion of several noxious agents evoking a reaction of the arterial wall There is nothing specific about this reaction, the wall goes through a stage of increased permeability, edema, intimal proliferation, medial fibrosis and adventitial vascularity The end result is either stenosis, obliteration or, rarely, aneurysm formation These physicochemical injuries are important because they may present surgical problems of revascularization and restoration of continuity But their study is also rewarding

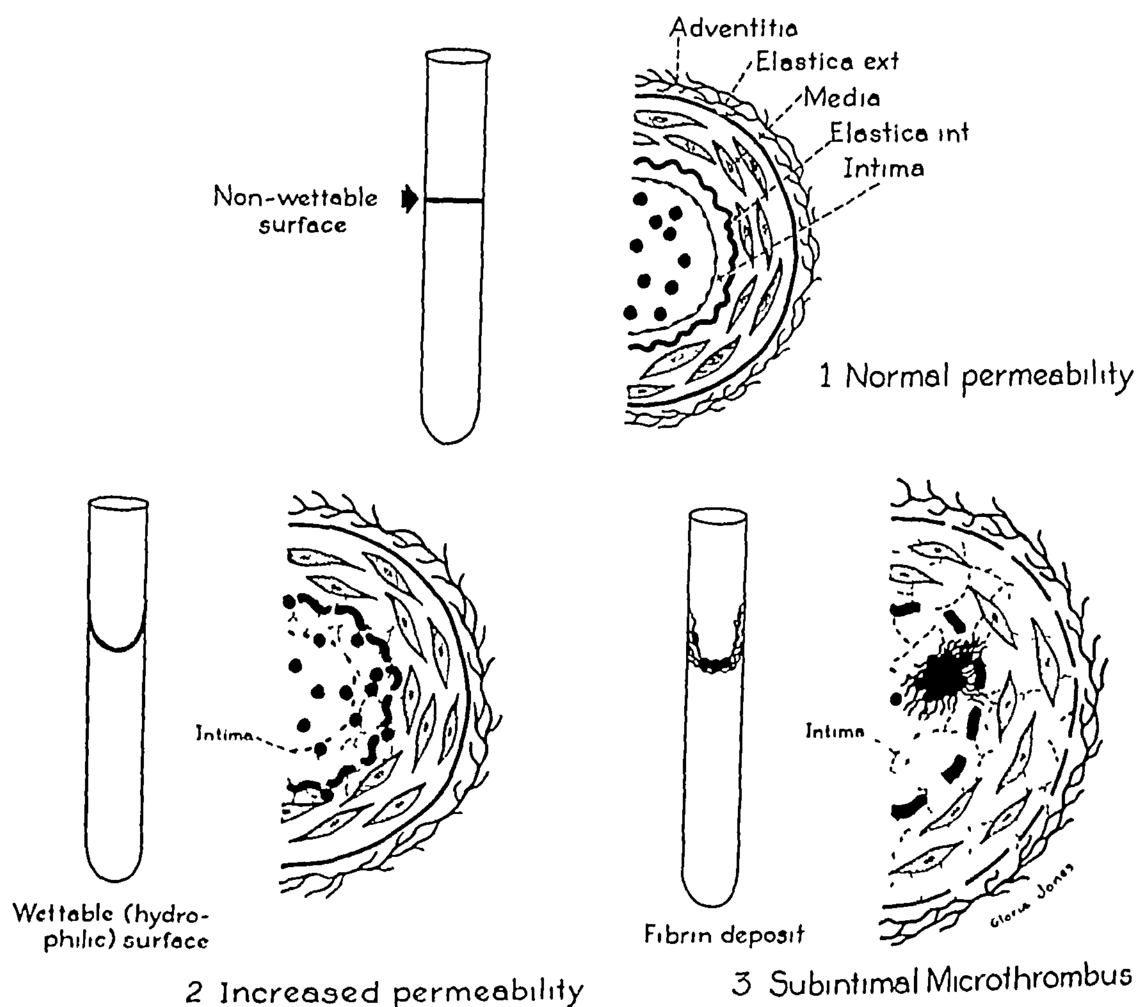


FIG 102 Permeability of the vessel wall Normally, there is a barrier between the constituents of the blood and the vessel wall The endothelial surface is nonwettable When the intima is injured it develops a wettable surface and cellular and plasmatic elements pass through it The internal elastic membrane becomes fragmented, and finally platelets and fibrin are deposited on the intima The wall imbibes plasma, becomes edematous and a plasma clot develops All this is a reaction of the wall to its change in permeability (Diagram modified from da Costa, J C · In Les Obliterations Arterielles Chroniques Trans European Section of Intern Soc of Angiology, Strasbourg, Oct 5-6, 1952)

since it leads to a better understanding of vascular reactions to infectious agents and even to the deposition of lipids as it occurs in atherosclerosis Re active repair seems to be the common denominator as a response to these injurious agents

That *endothelial permeability* is the cause of changes in the vessel wall was first emphasized by Schurmann and McMahon [21] They spoke of an interruption of a normal barrier between blood and vessel wall as a result of which edema proteins lipids calcium and red and white blood cells can enter the vessel wall be deposited there and evoke considerable reaction (fig. 102)

Deposition of hyalin and fibrinoid substance of lipids and calcium are commonly found in various vascular diseases Interestingly prolonged vaso-spasm produced by pituitrin results in extravasation of red cells in the wall of the artery proliferation of the endothelium and adhesiveness to particulate matter [22]

The point should be made that the duration and intensity of the stimulus are as important in determining the reaction of the vessel wall as the specific noxious agent It has been emphasized long ago by Gruber,[23] one of the keenest students of the pathology of frostbite that the histological picture of a vessel exposed to prolonged cold and the pattern seen in thromboangiitis obliterans are often indistinguishable This is mentioned only to discourage taking arterial biopsies at the slightest provocation which at best can only slow the various stages of response to injury infection or sensitization One is often impressed with the multiplicity of biopsies taken in indeterminate types of vascular disease and the indifferent yield which is obtained This is not to argue against the value of biopsies in certain situations especially in vascular lesions associated with collagen disease It must be clear however that in most teaching or general hospitals no Aschoff no Klemperer no Ewing nor Rich is going to report on the sections, and the healing stage of obliteration will give a nonspecific picture The reaction of the wall of the vessel to a multitude of noxious agents is a nonspecific attempt at repair [24]

Injuries Due to Heat

Vascular injuries due to burn are seldom discussed separately since all tissues are damaged Yet the local capillary injury resulting in increased permeability with regional loss of plasma into the tissues and 'weeping to the outside seems to be the initial factor resulting in hemoconcentration de creased blood volume decreased cardiac output, decreased blood flow sec ondary vasoconstriction and fall in blood pressure This is not the place to discuss the mechanism and management of burn shock. In the huge literature on burns however little attention has been given to the damage of major arteries and veins The marked stasis and hemoconcentration are predispos ing factors of thrombosis and immobilization of the burned area plays a part in this Many severely burned lower extremities exhibit deep venous thromboses and occasionally a thrombus of a major artery is seen even in

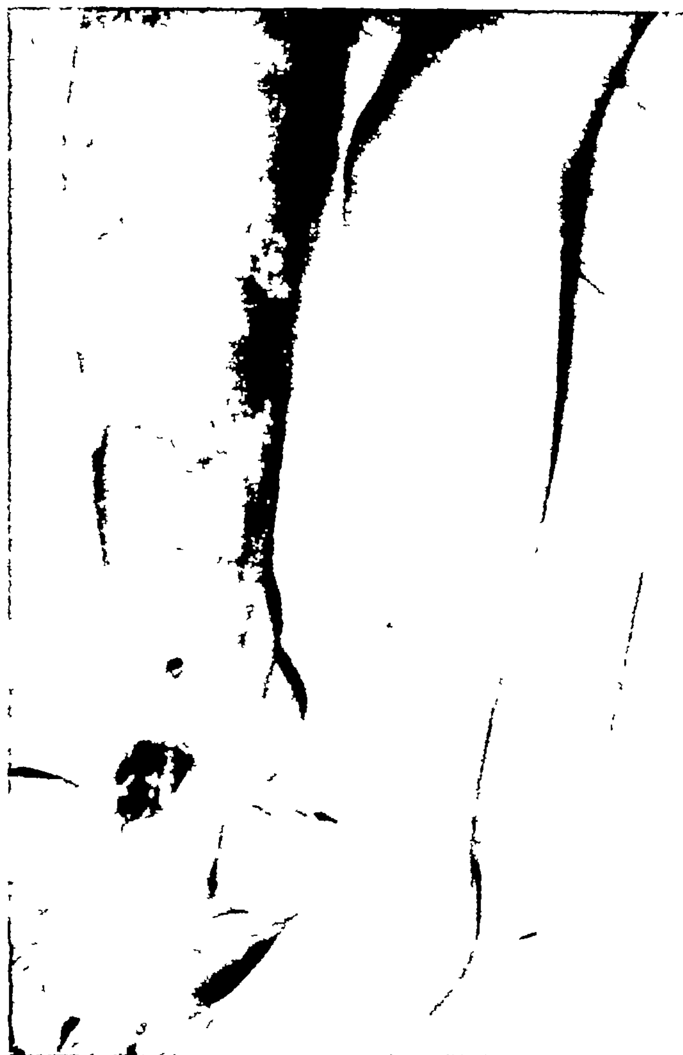


FIG 103

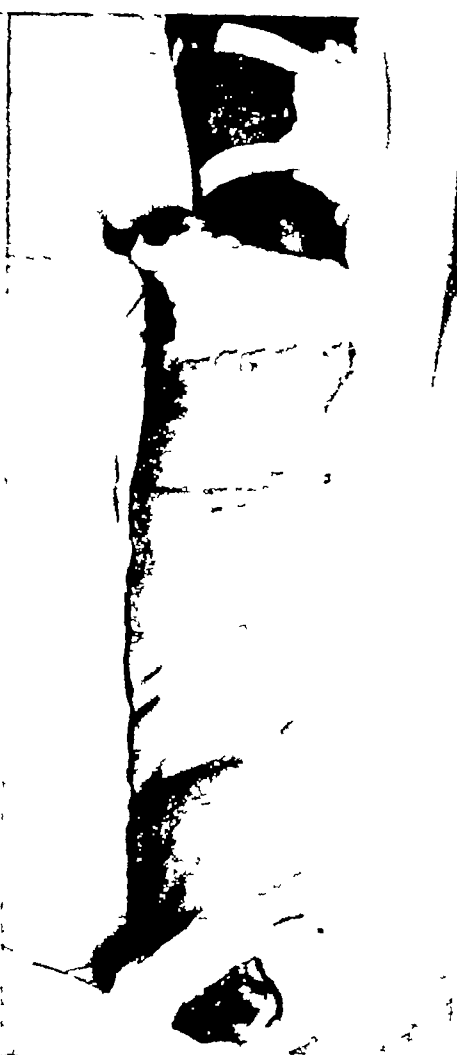


FIG 104

FIG 103 Typical burn in an ischemic limb to which a hot water bottle has been applied Patients often apply heat to a cold, numb extremity Physicians frequently advocate icebags

FIG 104 The ischemic extremity is best protected from external pressure, from changes in temperature and from frequent examinations by a large wrap of absorbent cotton, which prevents dissipation of heat and permits an air space between the skin and the *loose* dressing

young individuals in whom plasma loss and hemoconcentration have not been vigorously treated Heat produces vasodilation of the entire vascular tree, under certain conditions, however, excessive heat causes vasoconstriction, especially in the terminal vascular bed Of course, heat above ordinary room temperature is dangerous for ischemic limbs and the acute arterial embolus treated by an electric pad gives a typical picture (fig 103) The optimal temperature for an ischemic limb is body temperature or slightly below it, and wrapping the extremity in generous wads of absorbent cotton is a simple and highly useful procedure (fig 104); this is simpler and less expensive than heat cradles with controlled temperatures which were used on many services, including our own, 20 to 30 years ago

Heat stroke and heat exhaustion are not within the scope of this monograph

INJURIES DUE TO COLD

The problem of cold injury invariably comes to the fore during military campaigns undertaken in the winter There were about 55 000 patients with cold injuries from World War II and from 12,000 to 15 000 such patients from two winter campaigns in Korea Shumacker and Lempke[25] have given an extensive critical review of the recent advances in this field and made practical suggestions regarding treatment Since I was privileged to attend between 1943 and 1951 a number of meetings of the National Research Council devoted to the problem of cold injuries most of the material presented here is based on these informal discussions Also the *Follow-up Studies on Veterans with Cold Injuries** has given an unusual opportunity for studying the late effects of cold injuries

Certain facts are immediately apparent Cold injuries are rare in the civilian population on the other hand the mass of cases occurring during the war can seldom be studied carefully because of the necessity of evacuation from one medical installation to the other because of the scarcity of trained personnel and because of the inability of one individual or one group to follow through any of these injuries from exposure to final rehabilitation However just as in the case of vascular injuries the Korean theater of operations afforded an opportunity for an intensive study of early cases personal experience at the Great Lakes Naval Hospital with the late effects of cold exposure was equally instructive

Susceptibility to Cold Injury

Sensitivity to cold might be briefly discussed This can be distinguished from ordinary chilblains in that with sensitivity to cold there is a systemic as well as a local reaction to cold Urticaria develops on portions of the body directly exposed to cold air or water (fig 105) but there may be a generalized urticarial reaction angioneurotic edema, fall in blood pressure and rise in pulse rate Horton Brown and Roth[26] called attention to the fact that a number of unexplained drownings may be due to such a cold allergy in individuals who go into a shocklike state when exposed to cold water One of the most talented and popular surgeons of the Mayo Clinic has been said to have drowned this way † This peculiar phenomenon which may be due to a physical allergy formation of autoantigens histamine like substances or disturbances in the central heat regulation, has no bearing on the problem of cold injuries since they are overwhelmingly due—with the exception of high altitude frostbites—to prolonged exposure to cold In fact, this reaction may warn and protect the individual from continued exposure However a *high vasomotor tone* might truly predispose to cold injuries and suggestions have been made in the past to screen such individuals or to train them for cold adaptation.

* Cold Injury Project No 175 Committee on Veterans Medical Problems, National Research Council.

† Howard K. Gray a fine surgeon and true gentleman.

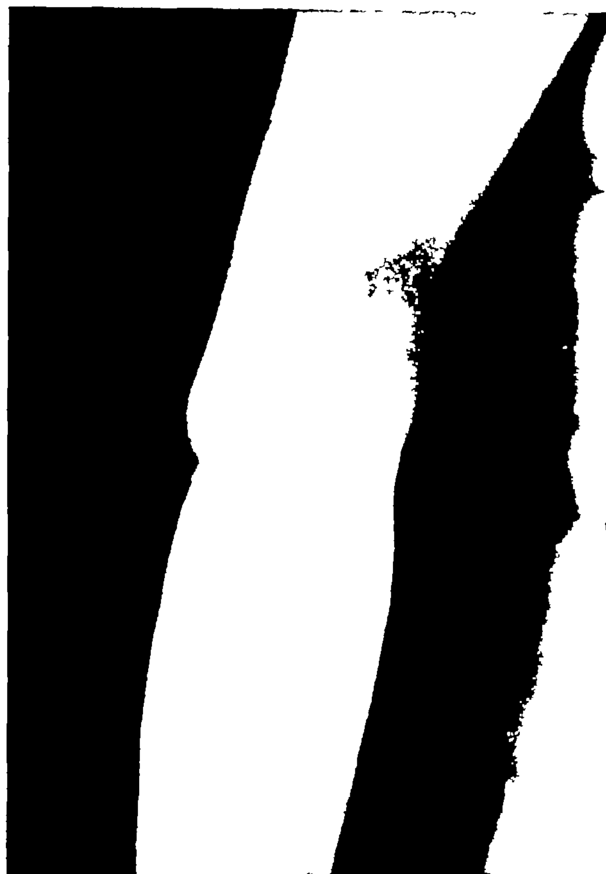


FIG 105 These giant wheals developed after a chip of ice was applied in two places This patient could never take a cold shower without fainting

Cold adaptation studies in the experimental animal and in man have been carried out in several centers In the human experiments, *ability to protect oneself against wet cold* is an important factor and requires thorough indoctrination, as carried out by the British for the Battle of the Bulge It is highly unlikely that armed personnel with high vasomotor tone could be exempted from exposure to cold, it is certain, however, that individuals who have suffered cold injuries are susceptible to recurrent damage and are preferably deployed to temperate or warm zones Their vasomotor regulation is definitely dampened *

Types of Cold Injury

Frostbite, trench foot and immersion limb are not always readily distinguished from one another Duration and intensity of the exposure, the amount of moisture surrounding the part, the vascular tone of the individual, his basal metabolism and his nutritional status, together with the effect of constricting shoes or garments, are some of the factors which decide the type of injury sustained It may well be that proximal to a typical frostbite a type of immersion limb injury exists The dry, freezing injury, forming crystals in the tissue which rupture the cells and are then thawed out by warming, may

* Cold Injury Project No 175 Committee on Veterans' Medical Problems, National Research Council

truly be different from a nonfreezing injury causing vasoconstriction capil lary permeability and tissue anoxia -[7] For clinical purposes however we recognize frostbite immersion limb (or trench foot) and chilblains

FROSTBITE With the exception of high altitude frostbite of airmen [78] which is brought on by a short exposure to low temperatures of minus 40 to 52 C and produces a true freezing of tissue most frostbites are due to prolonged exposure to moderate cold with contributing factors of dampness wind constricting clothing and pre-existing arterial disease Differentiation between frostbite and trench foot is not always clear or possible

That a severely injured man who is also suffering from malnutrition blood loss hypotension and immobility will suffer more severely need hardly be emphasized

IMMERSION LIMB OR TRENCH FOOT In *trench foot* the exposure is to higher temperature than that causing frostbite In the trenches the soldier is immobilized for 24 hours or longer and is hungry terrified and exposed to slush or wet mud with temperatures at or slightly above freezing. *Immer sion limb* is the result of prolonged immersion in water of an immobilized constricted extremity as for instance, in individuals who had to spend days or weeks on life boats or rafts as a result of shipwreck Most of the cases re ported have occurred in cold areas in the North Atlantic although James C White [9] has described them as occurring in comparatively warm water While records of trench foot go back to the earliest times in the early months of World War II the terms water bite life boat leg and sea



FIG 106 A purplish painful induration in a poliomyelitic limb was excised following a lumbar sympathectomy There is much fat necrosis, scarring and cuffing of small arteries, indicating perivascular damage due to increased permeability This 34 year old patient showed no other evidence of vascular disease.

boot foot" were used. Trench foot and immersion limb are probably identical, immersion limb has been described in Royal Air Force personnel who have spent many hours on exposed Scottish hillsides after airplane crashes.[30] The temperature does not have to drop below freezing, but dampness and windchill are prominent

CHILBLAINS Acute chilblains predominently affect the hands, feet and legs of bobby-soxers, many of whom wear thin hose or no hose at all, are anemic or hypometabolic Chronic chilblains occur on repeated exposure Poliomyelitic and obese, lipedematous legs are prone to develop large, purplish, sometimes itching indurations, often mislabeled as erythema induratum and suspected to be of tuberculous origin Actually, acute chilblains are the earliest state of a localized acute frostbite, seen more recently in patients under hypothermic anesthesia, chronic chilblains may be considered as a mild chronic frostbite Often called pernio, chilblain is a vascular disease and not a dermatologic one[31] (fig 106)

Functional Pathology

The discussion of cold injuries in a monograph on vascular surgery is not out of place because of their important vascular component Nevertheless, a direct tissue damage due to freezing and thawing also occurs, with the formation of crystals and rupture of cells, a true cellular injury

Man's sensitivity to mild and moderate cold contrasts greatly with the resistance to severe cold of hibernating or arctic animals [32] The white fox sleeps on snow or at atmospheric temperatures as low as 50° C. without experiencing frostbite or even shivering Pigeons seen in the arctic survived 144 hours at minus 40° C , but died within 20 minutes if exposed to the same temperature after their feathers were plucked Insulation and increased metabolic rate, however, are not the only factors involved The legs and feet of birds and of arctic mammals endure long periods of temperatures which would be harmful to human tissues Irving[33] brought forth some evidence of biochemical adaptation of the fats and other components in the exposed parts of these animals.

In man, severe vasoconstriction occurs on exposure of an extremity to severe cold According to Sir Thomas Lewis, three reactions are involved [34] First there is a local and direct effect of cold on the blood vessels, independent of nervous control Second there is a reflex vasoconstriction mediated by the sympathetic nervous system, and third there is a generalized and persistent vasoconstrictor response originating from the vasomotor center which reacts to the cooling effect of blood. All these reactions are primarily heat conserving mechanisms but as a result of severe and long-lasting exposure, tissue anoxia develops Two factors mitigating anoxia are the failure of the blood to part with oxygen at low temperatures and the opening of the arteriovenous shunts

When these physiologic defenses fail, freezing occurs During freezing

no blood flows through the frozen part and what is clinically significant, vasoconstriction is demonstrable proximal to the frozen part as shown by Shumacker and his co-workers [35]

Thawing brings on a set of reactions which resemble reactive hyperemia, aseptic inflammation or a histamine like reaction it tends to occur along blood vessels The skin may remain blanched and numb for some time but, all of a sudden, the skin becomes hot and red the pulses are bounding and severe burning pain develops Even during this time the major arteries proximal to the lesion may remain constricted With the return of blood flow edema begins to form because the capillary wall has become permeable After severe frostbite the edema fluid is rich in protein and may be hemorrhagic. If the frozen area is large, plasma loss and hemoconcentration develop just as in burns

Coincident with the formation of edema capillary stasis, agglutination and sludging of red cells is seen all of which almost completely stop the flow of blood, at least in the terminal vessels According to Friedman Lange and Weiner [36] heparinization prohibits such agglutinative thrombi, a statement which was later challenged by Laufman, Martin and Tanturi in their paper on the phenomenon of sludge [37]

After stasis has developed blood continues to flow through the arteriovenous shunts but capillary circulation is interrupted Thrombosis supervenes in many of the terminal vessels the frostbitten part blisters and exudes edema fluid When gangrene occurs, it is first wet poorly demarcated and often secondarily infected The extremity which escapes gangrene may show atrophy and marked fibrosis The protein rich edema organizes the fat becomes necrotic Muscle may often become necrotic, even when the skin remains intact, and R. B Lewis and his associates have been especially emphatic about the primary cause of muscle necrosis being, not ischemia but a direct effect of cold on the tissue cells [38] The small nonmedullated vasoconstrictor fibers sometimes survive when other types of nerve fibers are injured An obliterative endoangiitis, hardly distinguishable from a late stage of thromboangiitis obliterans, and marked sympathetic hyperactivity are often present.

Grades of Cold Injury

As in burns one can distinguish between four grades of cold injury Grade 1 is hyperemia without blistering grade 2 is hyperemia with vesicle formation grade 3 is necrosis of the skin and subcutaneous tissue and grade 4 is deep necrosis of the skin and loss of tissue

It should be emphasized that when seen early the pale wrinkled numb skin gives no indication of the extent of damage The success of therapeutic measures, whether rapid thawing, sympathetic block or indirect heating, will primarily depend on the extent of injury Devitalized necrotic tissue cannot be revived but the reactive edema around the necrotic focus is reversible

Late Sequelae of Cold Injury

When the dead tissue has been removed or has spontaneously sloughed off, or when the skin has regenerated after having been blistered, there remain the digits, earlobes, nose or other exposed areas which are sensitive to cold and which itch or burn on exposure to cold A neuropathy develops not unlike that in causalgia, where vasoconstrictor and sweat fibers remain intact, some of the somatic sensory fibers are demyelinated so that cross stimulation occurs (see causalgic states, p. 421) In addition, a fibrosis of the subcutaneous tissues and of joint capsules and tendon sheaths develops as a result of the plasma exudate and its clotting Muscles may also show necrotic foci and fibrosis That an emotional overlay and possibly a "compensation neurosis" also appear is well known to all who had to examine a large group of veterans with cold injuries Here the personal traits of the patient as revealed by the Cornell Medical Index[39] or by other forms of interview are of great help

Prophylactic Measures

In civilian life most cold injuries can be prevented; during war and especially in combat the intensity and duration of exposure, the necessity of immobilization in trenches, in cold water or in the snow, the associated injuries causing hemorrhage, hypotension with vasoconstriction and last but not least the mental strain all constitute factors beyond control

Clothing should be loose, light and warm, the insulation provided by "dead air space" between layers of clothing and the skin is most important. The effect of wind is well known and even the "internal wind" caused by movements of the wearer can lower the insulation of the clothing However, this disadvantage is overcome by increased heat production of exercising muscles Where sweating is apt to occur, as on the soles of the feet in a man under emotional tension or in battle, adequate vapor permeability is more comfortable, even although keeping the feet and socks dry by repeated changing of socks and by use of foot powder are urged A so-called "barrier rubber sock" and the insulated rubber combat boot may measurably decrease sweat production While ordinarily it is regarded deleterious to lose body heat by evaporation, it may well be that the condensation of vapor in the clothing raises the temperature there so that the gradient of temperature from skin to clothing is less than it would otherwise be [40] The last word as to the use of boots with rubber soles or leather soles with vapor permeability in dry arctic cold has not yet been said In water, mud or snow, insulation from moisture is important

Treatment of Cold Injury

The description of the functional pathology of frostbite (p 150), should serves as basis for the therapeutic attempts directed against cold injuries

FROSTBITE Human frostbite is a slow gradual process occurring over a period of hours in an individual with high vasomotor tone A quickly frozen rat s tail or rabbit s limb is not the equivalent of human cold injury Nevertheless, a great deal has been learned through well controlled animal experiments as carried out by Shumacker and his associates [25]

The routine use of antibiotics tincture of Zephiran chloride 1 1000 booster doses of tetanus toxoid in open lesions, strict bed rest and high caloric and high vitamin diets have proved their value at the Osaka Cold Treatment Center in Japan Early quick warming although experimentally useful has proved to be extremely painful The unanesthetized rabbit s ear does not tolerate rapid thawing in baths of 45 to 50 C [41] It may be how ever that the immediate rapid warming in water of 42 C which is only continued until the extremity has become thawed and warm is so important that intravenous short lasting anesthesia would be warranted At the present time since this treatment is still under trial a large cotton wrap placed around the frostbitten extremity and a room temperature not exceeding 72 F seem the safest procedure Rubbing with snow cold packs and cool baths the so-called slow warming of the frostbitten part are to be discouraged

One should be skeptical of the use of pressure dressings, plaster casts antihistaminic drugs rutin and heparin The so-called frostbite cocktail con sisting of alcohol procaine and heparin in 5 per cent glucose has also been abandoned

Priscoline and hexamethonium have been used in one of the army centers and while significant rises of skin temperature have been obtained there was no evidence that any tissue damage had been averted The same was true of ACTH and cortisone

Considerable controversy exists about the use and timing of sympa thetic blocks and of sympathectomy In the first stage of massive vasocon striction I have observed a rapid warming and flushing of a pale numb stiff ear Stellate block was done a few hours after prolonged exposure and no tissue damage resulted. In another instance, for the frostbitten feet of young boys who escaped from a reformatory school and hid in a barn with tempera tures around 0 F sympathetic blocks and sympathectomies were done to hasten demarcation and limit the spread of wet gangrene While digits were lost proximal edema and vasoconstriction were eliminated The most con vincing case of the value of early blocks and sympathectomy in frostbite is that of a young Negro woman who lay inebriated in snow for six hours and whose cold injuries affecting the feet were symmetrical and identical. One side was sympathectomized, the other one was not The edema on the dorsum of the foot disappeared and the demarcation of mummified gangrenous toes occurred in one week, whereas the control side exhibited the same picture six weeks later (fig 107)

Our very limited experience with sympathetic denervation in civilian frostbites leads me to believe that, depending on the degree of frostbite, vari ous degrees of improvement can be expected. In a first and second degree injury edema and vesicle formation can be quickly abolished whether cold



FIG 107 Fourth degree frostbite due to exposure in deep snow for six hours Damage seemed identical on both sides and midmetatarsal amputations were necessary after demarcation On the right, sympathectomized side, demarcation, loss of edema and mummification developed in one week It took the opposite, nonoperated side six weeks to reach the same stage

sensitivity remains has still to be determined In the irreversible tissue damage of grades 3 and 4, proximal edema and vasoconstriction are eliminated and demarcation is hastened Thus tissue damage is not minimized but earlier definitive treatment with shortening of hospital stay is accomplished

These observations are at variance with the opinion of many men whose experimental and war experiences are extensive First of all, it has been stated that during the period of reactive hyperemia maximal vasodilation is already present and sympathetic block in this phase increases edema and increases sloughing In spite of the marked hyperemia, however, the vascular tree proximal to the cold damage is spastic Sympathetic block in inflammatory edema, such as occurs in acute periarthritis, is anything but harmful

In the early phase of vasoconstriction and the late phase of cold sensitivity, hyperhidrosis and cyanosis, sympathetic block or sympathectomy is useful In this third phase of high vascular tone, neuropathy will not be benefited and burning paresthesia due to a partial nerve damage may even be aggravated At the Veterans' Administration Hospital at Hines, Illinois, a distinction has always been made between the overwhelmingly vasospastic

as against the neuropathic sequelae of cold injury In the latter, one can easily aggravate the burning paresthesia The same problem is encountered in the treatment of diabetic neuropathies with sympathectomy When the nerve lesion is on an ischemic basis, pain relief may be definite The question of sympathetic denervation in partial nerve injuries will be discussed with causalgic states (p 447)

Everyone agrees that the most rewarding avenue of approach is prophylaxis After exposure to cold has taken place, the object of treatment is not to do more harm than already has occurred

IMMERSION LIMB OR TRENCH FOOT These variations of cold injury will be discussed very briefly since they only vary from frostbite in the long duration of relatively mild exposure to cold in a damp or wet environment. The milder nonfreezing injuries in a wet environment do not cause deep necrosis unless pressure constriction or infection supervenes However the painful sequelae which are partly due to increased vasomotor and sudomotor activity and partly to somatic nerve damage mostly demyelinization of myelinated nerve fibers constitute one of the most difficult tasks to manage

The greatest concentration of immersion limbs during World War II was observed and reported by Webster and his associates in Halifax [42] They packed their patients limbs in ice and kept them cold for days slowly rewarming them and keeping the edema down in the second hyperemic phase

The argument here about quick rewarming and delayed rewarming is far from being settled In the volume *German Aviation Medicine in World War II* Siegmund[43] has laid down the general rule rewarm the chilled body rapidly rewarm the cold injured extremity slowly The Russian experience is to rewarm the extremities rapidly whereas the Swedes advocate rewarming the part rapidly then cooling it again

The immersion limb just as a trench foot or a chilblain goes through a prehyperemic hyperemic and posthyperemic stage While the feet are painless in the prehyperemic stage—in fact they are numb and pale—the hyperemic phase is exceedingly painful The hot, throbbing pain is reminiscent of the third phase of Raynaud s phenomenon but, of course it lasts much longer and is distributed over a much larger area When fully developed, the hyperemia follows no pattern of nerve distribution but bears a striking relation to the level of immersion or to the most proximal level at which color or temperature changes were noted immediately after rescue [44] The hyperemic phase may last from 6 to 10 weeks then almost imperceptibly passes into the post hyperemic stage

In the third phase where the swelling and hyperemia subside wasting of the intrinsic muscles of the foot is noted and a hollowing of the sole and marked clawing of the toes occurs The wasted muscle exhibits diminished electrical excitability Sir Thomas Lewis spoke of an algid stage The extremity is now cold cyanotic and moist with periods of excessive sweating This hyperhidrosis resembles that of causalgia and is accompanied by evidence of neuropathy There are patches of hyperesthesia, tingling, burning and sensitivity to cold An osteoporosis may develop in spite of continuous

weight bearing In severe cases this phase persists for a very long time In the case of a trench foot acquired in 1914 on the Austro-Russian front, I observed vasomotor and sudomotor phenomena with paresthesia and pain in the heel 30 years after exposure

CHILBLAINS These are nothing else but patchy, localized exposures to cold, and they go through the same three phases that any other cold injury does An acute and chronic type are distinguishable

Certain persons and certain areas are predisposed to chilblains Acute chilblains affect the feet, hands and legs of some adolescent girls, these girls are often anemic, hypotensive and have low basal metabolic rates Chronic chilblains produce discoloration, formation of nodules and recurrent ulceration on the calves Fat limbs of girls with the hypopituitary type of legs, and poliomyelitic extremities with atrophic, paralyzed muscles show patches of cold itchy cyanosis These reddish-blue patches have been known as Bazin's disease, erythema induratum, dermatitis hiemalis, lupus pernio and erythrocyanosis supramalleolaris They may clear up completely in the summer and get much worse in the winter In October or November small nodules occur which break down and form painful ulcers McGovern and Wright[45] have studied the histologic picture of these lesions and found an angiitis with intimal proliferation and perivascular infiltration, fat necrosis and chronic inflammatory reaction of the subcutaneous tissue in which giant cells are frequent No acid-fast bacteria or tubercles were found, although French dermatologists have often spoken of tuberculids The lesion is unquestionably a mild localized frostbite with angiospasm, anoxia, vascular permeability, fat necrosis and connective tissue repair It is a disease of bobby-soxers in a cold climate The German or Scandinavian Hausfrau with thick black woollen stockings does not get it

We have done a number of lumbar sympathectomies for this lesion, particularly in poliomyelitic limbs The plumb-colored lesions warm up, fade and the ulcers heal. Above all, cold sensitivity is greatly improved The lesion does recur, however, if the diagnosis of chilblains is incorrect Erythema induratum, nodosum and nodular vasculitis will not improve on sympathectomy Recently, Lynn[46] reported excellent results with lumbar sympathectomy for chilblains

CHEMICAL INJURIES

Certain toxic substances, notably lead, arsenic and mercury, are known to produce vascular phenomena from spasm to obliteration Their importance for the surgeon lies in the fact that the recognition of such an etiologic factor may lead to its elimination, in addition to the use of whatever measures are necessary to overcome the circulatory embarrassment In an early article (Mackenzie and de Takats, 1934[47]) we published data on three patients with lead poisoning and one patient with arsenic poisoning The patients with exposure to lead and increased lead excretion in the urine exhibited numbness, tingling vasospasm and obliterating endarteritis A patient with 5 mg of

arsenic in 1 000 cc of urine, who had calf muscle spasm on walking together with numbness and tingling, discontinued the use of a spray containing arsenic with physiotherapy and exercises she became free of symptoms During prohibition the moonshine often contained lead and arsenic and some of the drinking water contained unusual amounts of lead

In a group of six patients, all suffering from Raynaud s phenomenon an increased excretion of arsenic was found following the injection of sodium thiosulphate [48] A most striking case of livedo reticularis was seen in an infant in the wards of the Research and Educational Hospitals of the University of Illinois whose mother ingested and died of arsenic poisoning the baby was delivered by cesarean section

The renal arterial and arteriolar changes of chronic lead poisoning are sometimes associated with similar lesions in the arteries of the brain the retinal vessels the intraneural vessels in polyneuritis or in the arteries of the stomach Kada thought that lead poisoning was present in 3 per cent of all cases of spontaneous" gangrene of the extremities [49] although the experimental production of these lesions is usually unsuccessful

There are probably many other poisons which affect the vascular tree As pointed out in 1935 the vascular tree responds to many forms of injury by endothelial proliferation cushions of cellular masses under the intima medial edema perivascular reactions and perineuritis [50] The subject is only mentioned here to be considered in the differential diagnosis of atypical forms of vasospasm and endarteritis

Electrical Injuries

Our knowledge regarding electrical injuries of the vascular tree stems essentially from two sources First is the overwhelming clinical experience of Jellinek in Vienna who examined 4 000 patients exposed to high voltage electric current and who established a Museum of Electropathology at the University of Vienna [51] Equally important is the work of Langworthy and Kouwenhoven [52] who have reviewed the experimental data and contributed much to the subject

Blood pressure may rise to a very high level during the contact, followed by hypotension Convulsions and tetanic contraction of muscles may produce tendinous, fascial or joint damage Petechial hemorrhages in the central nervous system degeneration of peripheral nerves and demyelinization may occur

Blood is a splendid conductor and much of the current travels along blood vessels The wall becomes friable and endothelial damage medial rupture and mural thrombi are found

Felix Pearl has been a student of electric injuries in this country and the following statements are taken from his writings [53]

Contrary to the general impression high voltage currents are less dangerous than low ones so is high amperage usually less harmful than low Alternating current is 3 to 5 times more dangerous than direct The amperage

(voltage resistance) is difficult to determine because of the variable resistance of the body, and the resistance depends to a great extent on that of the skin Thus a current of 100 volts with a wet skin (1,000 ohms of resistance) could be fatal In a calloused palm the resistance may reach 1,000,000 ohms, but perspiration again will decrease resistance

The contact burns are round or oval and show no inflammatory reactions, but the resulting slough may cause massive hemorrhage weeks later because of the disintegration of the media of arteries far distant from the original lesion

Gas gangrene and severe delayed hemorrhage as a result of arterial necrosis have been seen by Gordin [54] If the patient is not electrocuted or if ventricular fibrillation and cardiac standstill can be relieved by cardiac massage, he may escape with some burns, a number of vague symptoms which are difficult to evaluate and an increased vasomotor reactivity which is not unlike the residue of an immersion limb Such a patient has recently been seen; he was exposed to a 3,000 volt circuit and, in addition to "neuralgic" pains in the right arm, he exhibited a cold, moist extremity with maintained pulses The temperature change could be abolished by paravertebral block A mild, partial nerve injury, as seen in causalgic states, seemed to be present

Recently, much interest has been aroused by the production of thrombi in normal aortas of dogs by creating small differences in the electric potentials across their walls [55] The thrombi form near the positive pole Avoidance of such microcurrents in vessel grafts may be one of the advantages of using lyophilized, nonliving arterial transplants or inert substances

Radiation Injuries

The effects of ionizing radiation on vessels have been reviewed by Shields Warren [56] The endothelium is the most sensitive part of the vessel wall Therefore, smaller vessels having large endothelial components show the greatest changes following radiation Endothelial proliferation with narrowing or obliteration of the lumina and thrombi are observed in the smaller vessels Larger arteries, however, seldom show injury when doses of irradiation are less than 500 r When 1,200 r doses are exceeded, the larger arteries show degeneration of their elastic lamellae, replacement of smooth muscle cells by connective tissue and intimal thickening

When young mice were irradiated with 410 r (31 r/min), histologic changes were produced in the elastic arteries that were comparable to those of physiologic aging [57] The elastic lamellae were frayed with an increased number of interlamellar fibers and an increased amount of ground substance

High voltage cathode ray irradiation is being used to sterilize blood vessel grafts Machines of 3,000,000 volts or higher must be available This procedure produces drastic alterations of the irradiated tissues unless they are in a frozen state during the entire period of irradiation [58] Less expensive irradiation of vessels for purposes of sterilization is the use of gamma rays.

) 000 to 3 000 000 roentgen equivalent physical units can be delivered out seeming harm through cobalt tubes [59]

The treatment of radium and roentgen ray burns forms a large part of ictivities of plastic surgeons The histologic study of excised ulcerations scars yield ample evidence for the destructive influence of radiation on d vessels The burned area must always be widely excised so that the ırteritis in the marginal zone does not interfere with the taking of skin ts

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CHAPTER 9

ARTERIAL INFLAMMATION

ARTERIAL INFLAMMATION, JUST AS ARTERIAL INJURY, SETS UP A SERIES OF REACtions in the arterial wall which may have some recognizable characteristics in the early phase of the disease but which terminate in nonspecific scarring with thrombosis, an endarteritis obliterans To further complicate the diagnosis, an arterial clot, whether thrombus or embolus, also sets up a response in the wall, whose nourishment now has to be supplied through the adventitia.

In addition to *bacterial* and *viral* arteritis, a sensitization of the vascular wall to bacterial and nonbacterial allergens produces a *hyperergic inflammation.* Thromboangiitis obliterans and periarteritis nodosa belong to this category. Our clinic has for many years treated Buerger's disease (thromboangiitis obliterans) on this principle and only regarded such lesions as being surgical when the acute lesion had subsided and when arterial obliteration was the problem.

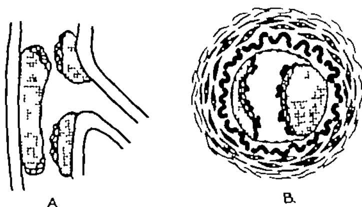
The uniformity of response to a noxious agent, be it physical, chemical, bacterial or allergic, make the intensity and duration of the stimulus as important as the nature of the stimulus For this reason, a detailed description of the individual types of arteritis is not as rewarding as the description of clinical forms under which they appear.

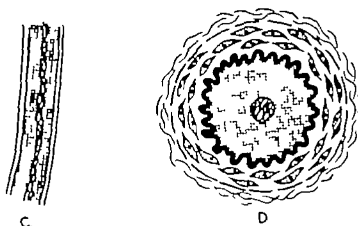
Clinically important entities will be first discussed These are thromboangiitis obliterans, periarteritis nodosa and temporal arteritis Next we will describe the rheumatic, tuberculous and luetic manifestations in the arterial wall and their end result as endarteritis obliterans Finally, mention must be made of a whole group of ill-defined vascular reactions, most of which belong to the collagen diseases and may at times have surgical implications.

CLINICAL FORMS OF ARTERITIS

1. THROMBOANGIITIS OBLITERANS

Thromboangiitis obliterans, while described many times before Buerger's publication in 1908, was really molded into an entity by this author and therefore justly bears his name Unfortunately, the diagnosis is made far too often and there are borderline cases and transitions into other clinical types of vascular disease. In this brief discussion I shall try to outline the pure form as it has appeared to us in our vascular clinics Whether or not





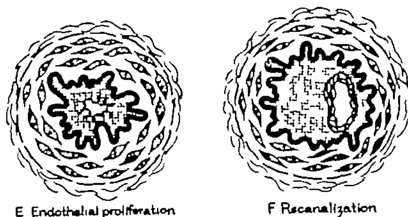


FIG 108 The stages of vascular occlusion in thromboangiitis obliterans (A) Subintimal cushions, typical of a vascular allergic response (B) Cross section of the same phase, showing clumps of platelets and fibrin over the reactive intima, edema of the wall and fragmentation of the internal elastic membrane. (C) A medium-sized artery is occluded with a fibrin plug. (D) The same phase in cross section. (E) The endothelium proliferates, leaving a small central lumen. (F) There is excentric recanalization. The lesion is in a quiescent state

its early appearance as "juvenile arteritis" predisposes to "presenile atheromatosis" is unsettled, but this important question will be discussed in a later chapter

Pathology

An excellent description of the progressive and recurrent pathology of thromboangiitis obliterans was given by Jager, and greatly influenced our thinking and our therapeutic attempts I am unaware of any improvement on his studies which were made almost a quarter of a century ago[1] (fig 108)

The lesions occur in segments, the artery and vein are matted together and often the accompanying nerve is encompassed by a scar One, of course, gets the impression that this is the organization of an exudate in and around the vascular sheath This segmental reaction is not specific for thromboangiitis obliterans since it occurs in panarteritis nodosa and other allergic reactions of the vascular tree The lesions begin often in the form of a segmental phlebitis with an acute leucocytic infiltration under the intima, the endothelial cells proliferate and are differentiated into epithelial cells and giant cells of the Langhans type Thus a tubercle-like picture develops, and we have observed the mistaken histologic diagnosis of a tuberculous phlebitis in cases of clinically typical thromboangiitis (fig 109) A little later, because of the endothelial defect, a thrombosis develops in the affected segment which is rapidly organized with a tendency to recanalize

In addition to these secondary thromboses there is a nonspecific proliferative endangiitis distal to the obstruction, which one sees in any vascular tree whose blood supply has been interrupted Leriche[2] spoke of ligature disease of the arterial tree, and produced endothelial proliferation, stenosis and obliteration of smaller arteries by a simple proximal ligature, depriving the endothelium from being adequately nourished from the lumen. I stress this because of the frequently negative histologic reports in patients whose clinical symptoms warrant the diagnosis of a thromboangiitis The site of the biopsy or amputated specimen is often *caudad* to the specific lesion and thus is nonrevealing Secondary infection in ulcerated or gangrenous toes often complicates the pure lesion For these reasons, the pathologist is seldom in the position to report a clear-cut positive diagnosis of thromboangiitis obliterans

Whether or not this primary inflammatory reaction will continue to progress to a fibrinoid necrosis and a secondary arteriosclerosis[3] will depend on the early recognition and intensive treatment of these exudative lesions When the internal elastic membrane is fragmented or destroyed, the cellular and plasmatic infiltration of the vessel wall may bring about all kinds of reactions of repair, none of which have any bearing on the original lesion

Predisposing Factors

The number of theories regarding the etiology of thromboangiitis obliterans is legion None of them have been proved and hence will not be dis-

cussed here in detail Certain clinical facts which can be occasionally rein forced by experimental studies do stand out, however and we shall list them as predisposing or contributing factors

AGE. Many authors have referred to this disease as juvenile arteritis which indicates that it is most commonly found between the ages of 20 and 40 This is peculiar since there is no obvious reason why a 50 year old individual when confronted with one of the etiologic factors would not develop the same vascular reaction The only answer one can possibly give is that the older age group has developed a decreased reactivity of the vessel wall to a noxious agent either because of immunity or because it is more rigid inelastic and incapable of much spasm This is obviously speculation but I have not found any other explanation for the selective localization of the malady in the younger group The male sex hormone active during this period certainly does not inhibit vascular reactions but on the contrary facilitates them (see experimental gangrene produced by ergot p 167) The adrenal cortex, with its diminished activity in the older age groups, may in fluence inflammatory reactions The sclerotic vessel may not respond so well to an inflammatory stimulus There seems to be no support for the idea however that hyperadrenalinism is responsible for the clinical picture and that adrenalectomy is a reasonable procedure as advocated for some cases by Leriche [2]

In our own material which comprises over 200 patients who were diag nosed as having Buerger s disease I am not aware of a single case in which the *onset of the disease* was above 45 years of age On the other hand patients who have been followed from an early onset into middle or old age



FIG 109 Tubercle like granulomatous inflammation in segmental phlebitis. This patient was a student in an Ozark school and was diagnosed as having tuberculous phlebitis. Cessation of smoking and adequate sodium intake arrested the lesion. A 20 year follow-up was made Note the infectious granuloma in a superficial vein of the calf with a Langhans-type of giant cell.

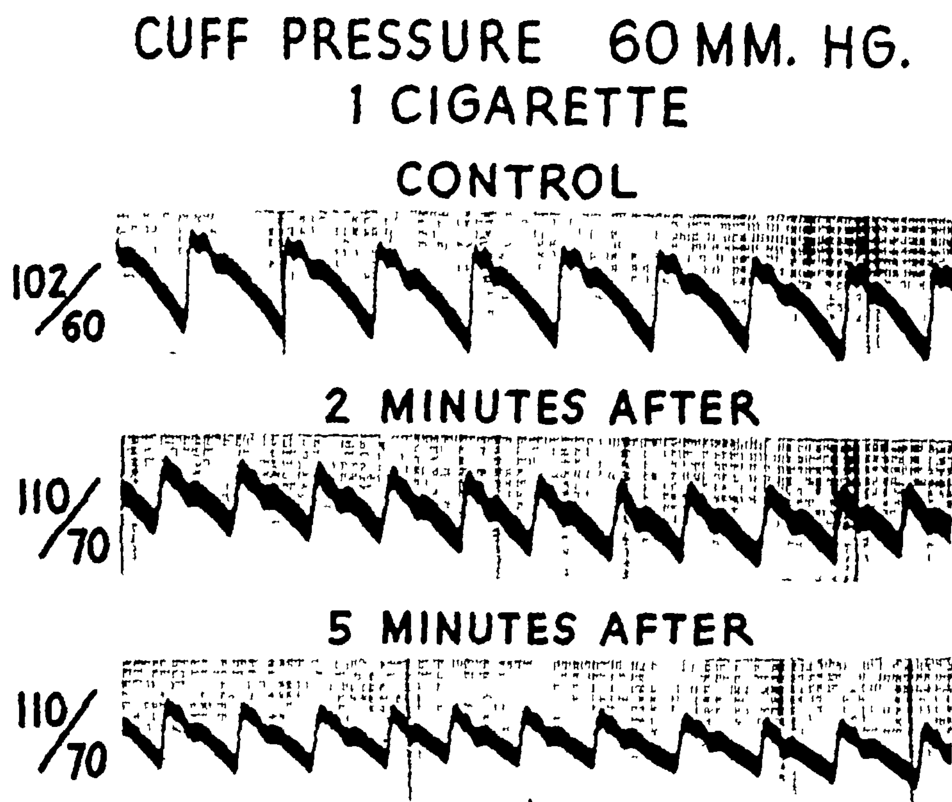


FIG 110 Recording oscillometric tracings of the effect of one cigarette on the pulse wave (Courtesy of Dr Carl A Johnson, St Luke's Hospital, Chicago)

have exhibited a picture of endangiitis obliterans with no acute exacerbations I am told that acute rheumatic polyarthritis does not flare up—as such—after middle age but simply goes into an ankylosing, noninflammatory state, a truly remarkable analogy

SEX In contrast to Raynaud's disease, which occurs approximately 70 per cent in females, thromboangiitis is seen mostly in males Seven women were studied out of the 200 patients in whom the diagnosis of thromboangiitis was made Every one of them was a heavy smoker and five out of the seven had certain male characteristics, which would suggest an hormonal imbalance We have not so far followed up this lead with urinary excretion of androsterones None of these women exhibited the fulminating course which is observed in a number of males, and all of them were readily controlled by the combination of medical and surgical measures to be described presently

TOBACCO Smoking causes vasoconstriction in the peripheral blood vessels The temperature of the fingertips shows a drop of several degrees after smoking one or two cigarettes, especially after deep inhalation. While some of this vasoconstriction is the reflex effect of deep inhalation, it is easy enough to show that the ischemia is overwhelmingly due to the nicotine and that smoking of filter paper cigarettes or cigarettes filled with chopped-up cigarette paper won't produce the drop in skin temperature [4] The decrease in blood flow after smoking can also be demonstrated by plethysmographs and recording oscillometers (fig 110) and it occurs as well after the use of "de-

nicotinized or mentholated cigarettes There is also a marked rise in blood sugar after smoking[5] which may be due to an epinephrine effect and which one does not find in patients with hypertension whose adrenal glands have been denervated.

Unquestionably however, some individuals particularly nonsmokers react more violently to tobacco than others perhaps because of increased vasomotor reactivity or because of an allergic response The latter mechanism has had several proponents starting with Harkavy s cutaneous tests [6] but generally speaking most authors have been unable to establish a specific relationship between thromboangiitis obliterans and tobacco allergy [7]

While smoking has a deleterious effect on the peripheral circulation of patients suffering from Buerger s disease and while no patient should ever be treated without observing complete abstinence from tobacco the available evidence only shows that it is harmful to smoke with thromboangiitis just as it is harmful for such a patient to injure his foot or to expose it to cold

ERGOT A number of patients first reported from Dr Buerger s clinic were Polish or Russian Jews whose daily diet contained black rye bread Ergot in Eastern Europe is an endemic parasite of rye and it has been known for many years that ergotism can produce gangrene

Outbreaks of epidemic ergotism still occur in modern times and a relatively recent report showed that the rye in Illinois contained ergot Ustilago was also found in corn [8] Actually ergot poisoning is only seen now when ergotamine tartrate is taken such as for migraine for the itching of jaundice or for postpartum hemorrhage

Ergot is a highly interesting drug because one can produce prolonged vessel spasm with it, leading to gangrene Thus McGrath[9] produced gangrene of the tail of rats Females could be protected from gangrene by large doses of Theelin the male rats could not.

Prolonged intake of ergot for purposes of weight reduction or control of migraine may produce numbness tingling, Raynaud s phenomenon and digital arterial occlusion. While it is well to eliminate this possible source of vascular disease with a careful history there is no evidence that ergotism may ever cause thromboangiitis obliterans In a case of our own, a middle aged pregnant woman was admitted to St Luke s Hospital with a cyanotic, pregangrenous foot of unknown etiology Only after a lower leg amputation did she reveal that she was a given a single, unknown dose of ergot for abor tion The leg was lost but the baby was carried to term Vitamin deficiency notably vitamin A deficiency is said to facilitate ergot poisoning.

FUNGUS INFECTION It is obvious that mycotic infection, and particu larly the superimposed bacterial invasion, is more apt to take hold in an ischemic area, and this is true in all forms of peripheral vascular disease In full blown cases of thromboangiitis obliterans, this complication is often detectable There may be however a closer etiologic relationship than this In young, vasospastic, hyperhidrotic individuals, one may observe acute spreading vesicular eruptions with ascending periphlebitis The arterial circu lation is not involved The periphlebitis which is actually a lymphangitis

there may still be no evidence of blockade of the major arterial pathways This of course does not mean that the subintimal cushions described by Jäger[1] are not present and are not narrowing the arterial tree As stated in a previous chapter however the lumen has to be stenosed to 60 or 65 per cent of its original diameter before the stenosis becomes clinically manifest The red streaks or nodules may affect any of the four extremities Fever or leucocytosis is usually absent Deep venous involvement does occur but most of the phlebitides are superficial Chilling, smoking, direct trauma or a veni puncture may light up a quiescent process Digital arteries have closed up in a violinist in a chiropractor and in a Florida tourist on whose foot a hurricane dropped a cocoanut The local lesions are hot throbbing and exceedingly painful but intermittent claudication is not a prominent symptom

(3) The process now takes on an accelerated course Toes or fingers become glossy and cyanotic edema develops Pulses of the foot or hand disappear The artery itself feels swollen and is tender to touch The skin is scaly and a dermatitis may develop which is readily sensitized to salves sulfanilamide derivatives or antibiotics often bring on generalized cutaneous manifestations

(4) The skin of toes or fingers ulcerate or patches of gangrene appear Osteomyelitis develops Pain is intractable, and since the patient hangs his feet down to get relief massive edema appears Heat carbolic petrolatum and minor surgical procedures accelerate the process Visceral lesions in the brain, heart lung, kidney and mesenteric arteries occur Coronary throm bosis or duodenal ulceration are not uncommon [12] Acute arterial occlusion of major pathways presents itself

(5) Spontaneously or as a result of adequate treatment, the process cools off and burns itself out with residual segmental venous or arterial oc clusions in various parts of the body In later years superimposed arteriosclerosis seems more advanced or a picture of endarteritis obliterans super venes

This course of events may not be followed in all cases This is obviously a schematic description of a progressive disease which may regress, either spontaneously or as a result of treatment may remain stationary or may go into remission or flare up Intensive therapy will halt the disease when first seen then the problem remains of preventing exacerbations and improving circulation around the segmental occlusion

Differential Diagnosis

Thromboangiitis obliterans is not a frequent vascular disease It has been frequently diagnosed in the presence of other peripheral vascular dis eases which will now be enumerated

ARTERIOSCLEROSIS OBLITERANS It is not unusual to find men below the age of 40 who either have atheromatous deposits in their blood vessels or who show medial calcification the latter picture being seldom respon sible for arterial obliteration Sclerosis of retinal vessels hypertension

hyperlipemia, diabetes or diminished sugar tolerance speak for presenile atheromatosis Spotty longitudinal calcification seen in the roentgen film speaks for atherosclerosis, whereas smooth, transverse calcified rings denote medial calcification Monckeberg's sclerosis In patients past 40 years of age, a thromboangiitis obliterans may well show superimposed atherosclerosis, but here the history of vascular insufficiency dates back to the twenties or thirties Closure of a radial artery or multiple involvement of digital arteries speaks strongly against arteriosclerosis, although occasionally it may occur On the other hand, "pulseless disease" of the whole arm is usually due to arteriosclerosis of the aortic arch (see chapter 18, The Aortic Arch Syndrome)

ARTERIAL EMBOLISM This may be subclinical or ignored at first because the clots are small, or arteries are affected whose occlusion is asymptomatic In patients suffering from mitral stenosis with auricular fibrillation, closure of pedal or radial pulses may be present without the patient being aware of it Only when another, major embolus is released from the heart is the embolism obvious The emboli may recanalize spontaneously or the occlusion is so well compensated by collateral circulation that only a moderate arterial insufficiency results with coldness, numbness, tingling and claudication In such a situation, I have seen thromboangiitis diagnosed. The history of acute onset, the presence of rheumatic heart disease, myocardial infarction, atheromatous plaques in the aorta or a subacute bacterial endocarditis lead one to think of an embolic origin. Again the patient may be unaware of any of these conditions, which can run an insidious, almost subclinical course

ARTERIAL THROMBOSIS There is an infrequent group of patients who develop a thrombotic occlusion of a seemingly intact artery The site of this may be in the popliteal artery, in the digital arteries, or almost anywhere else. The acute arterial thrombosis may occur after severe dehydration, after a marked hypotension or after certain types of septicemia, and there is usually a localizing factor such as an arterial stenosis or massive digital vasoconstriction to explain the site of occlusion Since arteriosclerosis and cardiac damage are not detectable, the diagnosis of thromboangiitis is readily made There is, however, no involvement of the veins and multiple segments are not occluded

RAYNAUD'S PHENOMENON Not only arteriosclerosis, but thromboangiitis obliterans, scleroderma and lupus erythematosus may show triphasic color changes Raynaud's phenomenon in males is frequently due to thromboangiitis obliterans, but a positive diagnosis will have to be based on other findings since the color changes are anything but specific

SECONDARY VASOSPASTIC PHENOMENA These may follow frostbite or accompany rheumatoid arthritis and many mechanical strains of the feet, such as pes planovalgus which is often associated with excessive sweating and coldness Also radicular pain originating from lumbosacral arthritis will produce pain on walking, cold feet and vasospasm A positive leg-raising test reveals many neuritic syndromes, and differentiates them from thrombo-

angiitis obliterans Reflex sympathetic dystrophy (minor causalgia) is readily differentiated from Buerger s disease All these syndromes will be discussed in more detail under their own headings Here I just wish to point out that they are often mistaken for thromboangiitis obliterans

The Spontaneous Course of the Disease

This segmental inflammatory reaction in arteries and veins begins as a rule in young men who sweat and smoke excessively and who exhibit the closure of one or several arterial or venous segments in the feet or hands Factors that seem to accelerate the disease are intercurrent infections exposure to cold and trauma In fact the pre-existence of a thromboangiitis obliterans is sometimes difficult to establish when these accelerating factors supervene

Later toes or fingers become painful ulcerate or show patches of gangrene (fig 111) Pedal or radial pulses disappear and claudication develops Generally speaking, the disease seems to spread from the periphery to the major vessels however there are exceptions to the rule Visceral vascular disease, which we have observed in the brain heart, kidney and mesenteric vessels, may be an early or late manifestation of the disease In a personal experience with visceral lesions in thromboangiitis obliterans [12] retinal le



FIG 111 This 42 year old man was unable to stop smoking and held an outside job with repeated trauma to his fingers. The lesion is acute, the fingers are swollen, the gangrene is moist. (Case of C.W Research and Education Hospital No 262 155 Jan 23 1942.)



FIG 112 Quadrilateral involvement in a 38 year old truck driver, whose lesions started insidiously five years before this picture was taken He had an abnormal cardiographic pattern, had suffered a temporary hemiplegia and was a complete invalid His further course is unknown

sions were found in 26 per cent of the cases, cerebral vascular lesions found in 10 per cent, abnormal electrocardiographic pattern found in 56 per cent, duodenal ulcer in 10 per cent, impaired renal function in 16 per cent, pulmonary fibrosis in 4 per cent and occlusion of the spermatic artery in 2 per cent The incidence of visceral lesions seems rather high in this small series It is probable that some of them are arteriosclerotic changes and are not necessarily due to thromboangiitis It is also true, however, that because of lack of routine study for generalized vascular disease, many of these visceral localizations are missed It is interesting that cerebral Buerger's disease may antedate, at least symptomatically, the peripheral vascular phenomena In three cases of our own series, neurologic manifestations occurred prior to the pulselessness of the feet A periphlebitis of the retina was seen in one of the patients, but this is the only one in whom we have ever observed this phenomenon

While visceral lesions may be rare in small series, most of the patients ultimately die of visceral thromboses

If the patient stops smoking and accelerating factors are kept at a minimum, the process may come to a halt and leave the patient with segmental vascular obstructions In other cases an almost violent progression takes place, leading to the loss of fingers or toes, or necessitating major amputations (fig 112) Many patients are unable to stop smoking, or return to smoking



FIG 113 Note the extensive sloughing of skin tendons and muscle this leg was unbelievably painful and neglected The popliteal artery was closed Meat and a magic salve smelling like carbolic acid were recovered from the dressing. (R.S., Reg No 155 743 V A Hospital, Hines, Ill., July 19 1945)

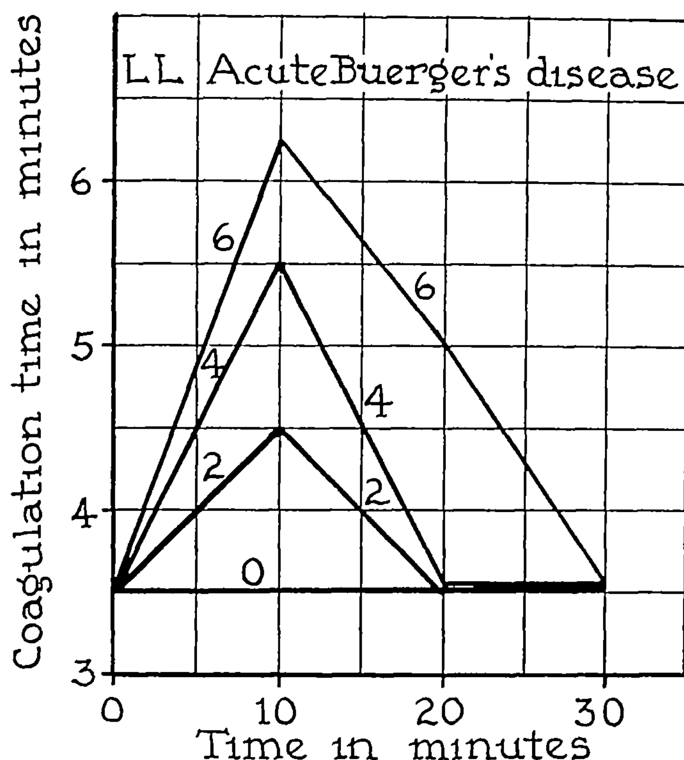
when the pain becomes unbearable They may become addicted to narcotics and their physicians may continue to treat painful and irreversibly lost parts with ointments vasodilators and pain killers Patients often seek advice at a time when they are demoralized from weeks and months of sleeplessness sitting up in a chair all night for relief of the ischemic pain creates a dependent edema and further interferes with circulation They may use electric pads hot towels and caustic salves to relieve the ischemic pain and thus produce incredible destruction (fig. 113)

The disease may be quiescent for a while and then be suddenly stirred up by trauma and smoking. Figure 112 shows the hands of a patient who had obtained a good remission following bilateral dorsal sympathectomy and amputation of digits Six years later he appeared as in this picture smoking again and having driven a laundry truck in severely cold weather

Thromboangiitis obliterans then runs an intermittent course with exacerbations and remissions Its treatment has to be carefully fitted to the stage of the disease (to its activity so to speak) and requires an understanding of its natural course

Treatment

THE EARLY INCIPIENT STAGE. The patient has recurrent attacks of segmental phlebitis on the dorsum of the foot in the plantar veins or in the forearm He may have one cold toe or digit Abstinence from tobacco is a must and this prescription holds for all other stages but it is difficult to enforce Only if the patient *knows* that he cannot get well while smoking, will he or she be able to stop [13] If ringworm infection is present, it should be



DAILY INJECTIONS OF SODIUM THIOSULPHATE

FIG 114 Heparin tolerance curves before and after injections of sodium thiosulphate Sulfanilamide will also effect an improved response Note the flat curve before injections Curves were obtained on the second, fourth and sixth day Capillary coagulation times were used (de Takats, G The Effect of Sulfur Compounds on Blood Clotting Surgery, *14* 661, 1943)

intensively treated by Desenex powder and ointment Soaks of potassium permanganate discolor the toes and nails and are messy If the blood count, hemoglobin or hematocrit is high, the patient is encouraged to drink ten glasses of liquids daily and use some salt tablets so as to get about 10 Gm (150 gr) of sodium chloride a day Naturally an "acid" ulcer of the stomach or hypertension constitutes a contraindication When the blood count is corrected this medication is stopped and the patient is usually found to be anemic Hypertonic sodium chloride injections, popularized in the East by Silbert,[14] have not been employed

Obvious foci of infection in teeth, tonsils and prostate are eliminated if possible A series of twelve injections of sodium thiosulphate in 1 Gm (15 gr.) doses is given intravenously within a period of four to six weeks Pain, the periphlebitis causing the red streak and further migration of the process to other vascular segments are usually stopped The rationale of treatment by sodium thiosulphate is not clear It may act as a general detoxifying agent, binding the toxin or allergen responsible for vascular reaction It may influence the clotting mechanism, since it increases the patient's response to heparin[15] (fig 114) It may so modify the ground substance of the vessel wall that the inflammatory reaction subsides

Whatever the mechanism, the acute process is remarkably influenced In the past, small doses of triple typhoid vaccine have been used in the treat-

ment of thromboangiitis avoiding chills and fever and simply creating a mild leucocytosis and a minimal rise in temperature These subreactionary doses vary between 100 000 and 1 000 000 bacteria and are cautiously raised avoiding chills and fever for a series of twelve injections [16] We have not used this method of late but it is far preferable to the large chill producing doses starting with 25 million bacteria and increasing up to 300 million It may act by increasing fibrinolysis

All these procedures are simply used to cool off the acute process that the process becomes quiescent may be ascertained by the diminution of pain and edema, by decrease in the sedimentation rate and by improvement in heparin tolerance Actually a four to six weeks course of sodium thiosulphate will usually bring on a remission

QUIESCENT STAGE WITH MULTIPLE ARTERIAL OCCLUSIONS (fig 115) The disease now is inactive but peripheral circulation especially the peripheral arterial tree is crippled by occlusions which produce numbness tingling, cold sensitivity and intermittent claudication In such a stage dorsal and lumbar sympathectomies are of great help and should be done without delay However depending on the state of the terminal vascular bed and in the face of a paradoxical drop of skin temperature after sympathetic block amputation of fingertips or toes may have to be combined with sympathectomy to effect a rapid rehabilitation of the patient One sees months and years of



FIG 115 This patient has no pain at rest he can sleep without sedatives but is unable to use his hands for any gainful occupation. Dorsal sympathectomies resulted in spontaneous separation of gangrenous patches and epithelialization was complete in four weeks.

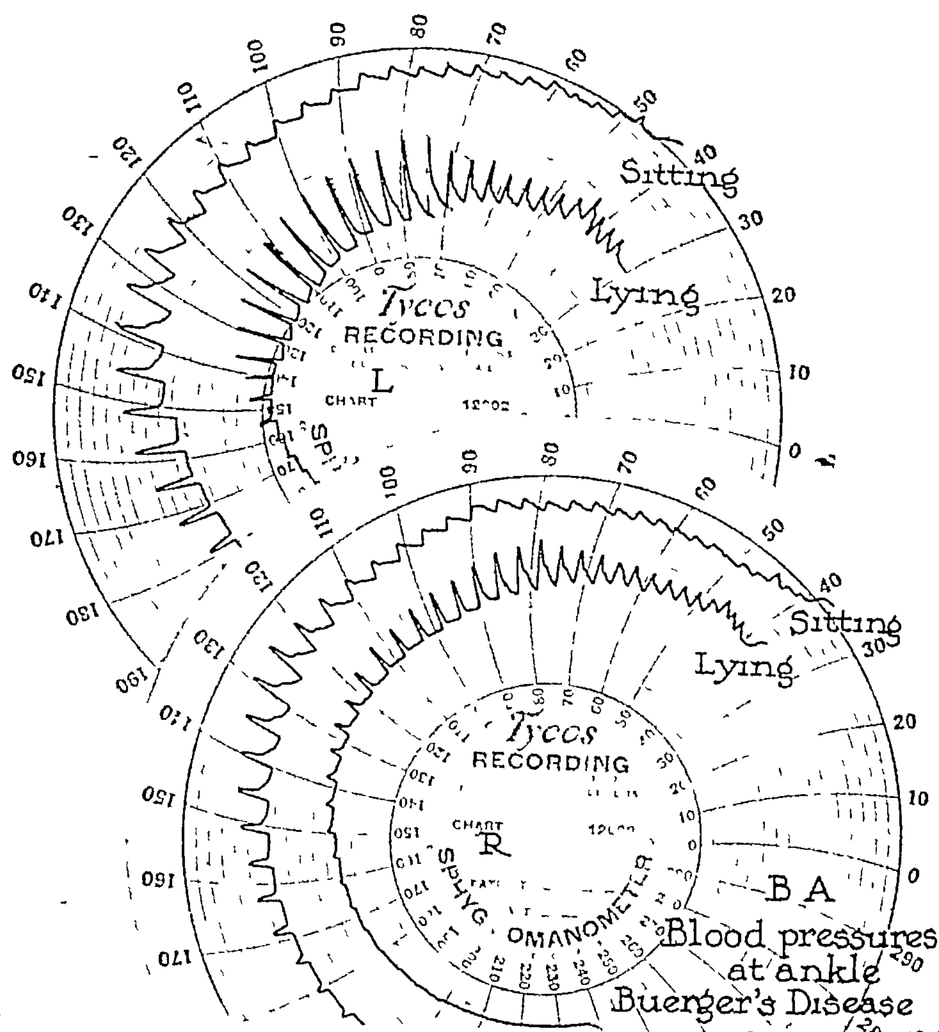


FIG 116 Oscillometric tracings at both ankles in a patient suffering from Buerger's disease With this patient, pulse waves could still be recorded since most of the vascular involvement was below the ankle Note that pulse waves are higher on the left than on the right side, and that hanging the feet down shifts the curve to the left because of increased vasoconstriction The tracings were made with a double cuff so that diastolic pressures are measurable These rose from 70 mm Hg to 120 mm Hg on both sides Such a postural response is abolished by sympathectomy (de Takats, Fowler, Jordan and Risley Sympathectomy in Treatment of Peripheral Vascular Sclerosis J A M A , *131* 495, 1946)

disability resulting from so-called "conservative" treatment of ulcerated or gangrenous digits Hanging the feet down over the edge of the bed may give temporary relief but increases edema and causes marked vasoconstriction with a localized hypertension in the dependent areas (fig 116) Toe amputations as high as through the proximal third of the metatarsus will give adequate walking surface Lower leg amputations should seldom be necessary but one is forced to perform them if a transmetatarsal amputation fails Syme's amputation, which gives surprising results in frostbite and arteriosclerosis, has not been employed in our clinic in cases of thromboangiitis, but it may find a limited indication

Arterial grafts in thromboangiitis have had very little success In one case communicated to us, a segmental resection and graft resulted in a flare-up of an acute process with thrombosis of the graft and the segment above it

and below it This is to be expected since even comparatively minor trauma can reactivate the disease Though subject to revision at present Buerger s disease constitutes a contraindication to arterial grafting.

CHRONIC, RECURRENT PHASE. The acute phase may recur after years of remission brought on by resumption of smoking, by exposure to cold by trauma and by intercurrent infection One does see an exacerbation in non smokers but this is rare Virus infections, such as those causing hepatitis and atypical pneumonia seem to have some affinity to the susceptible vascular tree in thromboangiitis obliterans The flare up of arteritis or phlebitis may occur in sympathectomized extremities but the patient tolerates these better and loss of tissue is not apt to be so great The treatment of this phase is identical with the treatment of the initial acute phase *i e* avoidance of vasoconstrictive influences, sodium thiosulphate injections and elastic support, particularly if deep venous involvement is present If the patient is showing superimposed arteriosclerosis the circulatory involvement is of course more severe In some patients whom I followed over a period of 20 years or more involvement of major arteries by atherosclerosis was not a prominent feature

TERMINAL, MALIGNANT PHASE In poorly treated neglected cases the



FIG 117 This patient had been free of any active disease for six years. His feet were warm, though pulseless, after bilateral lumbar sympathectomies. Following a bout of "flu (what virus?) he appeared with swollen infected hands. The dorsal sympathectomies done originally by an anterior approach were extended through transthoracic sympathectomies. Nevertheless, pain, ulceration and edema persisted. Anticoagulant therapy with heparin brought on a remission.

disease may suddenly appear in an accelerated form causing gangrene of multiple areas, such as all fingers or all toes (fig 117), and thromboses in visceral organs with a marked resistance to the effect of heparin Such patients require long hospital care, anticoagulant therapy and removal of nonviable parts at a level of circulatory efficiency They have had months and years of suffering, are demoralized and can seldom be rehabilitated They resemble the fulminating cases of periarteritis nodosa or lupus erythematosus and usually die of some superimposed infection in later years With earlier diagnosis and more intensive care one seldom sees this stage of the disease

Not used in any stage of thromboangiitis obliterans are the following popular procedures hypertonic salt solution given intravenously, Depropanex (muscle extract), oscillating bed, suction-pressure boot and Buerger's exercises An individual critique of these procedures would be out of place here Our present therapeutic aims are based on the assumption that thromboangiitis is a segmental, chronic, recurrent toxic or allergic reaction of the vessel wall After the disease becomes quiescent, sympathectomy removes the vasomotor component and ensures an even blood flow with improved cutaneous circulation Segmental excision and grafting of occluded arteries may reactivate the process The disease is primarily in the smaller and terminal arteries and arterioles

2 Periarteritis Nodosa

A group of diseases named "collagen diseases" may pose difficult diagnostic problems Of these, periarteritis nodosa (polyarteritis, panarteritis, necrotizing arteritis) shows primary localization in the vascular tree, notably a necrosis of the media, destruction of the internal elastic membrane and infiltration of the adventitia with polymorphonuclear leucocytes, lymphocytes and eosinophils [17]

Since the early symptoms may consist of vasospasm, numbness of fingers and toes, acute thromboses and small aneurysms (most of which, however, are microscopic), it is well to think of such a possibility in the differential diagnosis of peripheral vascular lesions A thorough discussion of this bizarre protean disease will not be attempted, since medical texts and the monograph of Allen, Barker and Hines[18] have given most adequate descriptions

The present trend is to regard periarteritis nodosa as an allergic reaction to drugs, an infectious process or a combination of them There is considerable overlap here between rheumatoid infection, thromboangiitis, lupus erythematosus and scleroderma, all of which show related pathologic pictures fitting into the category of hyperergic inflammation and resulting in fibrinoid necrosis.

Unfortunately, the typical histologic picture is seldom found in biopsy and the healed stage of periarteritis cannot be differentiated from other forms of arteritis *According to Arkin,[19] the organs most frequently involved are* the kidneys in 80 per cent of the cases, the heart in 70 per cent, the liver in 68 per cent, the gastrointestinal tract in 50 per cent, the pancreas

in 25 per cent the mesenteric artery in 30 per cent the muscles in 30 per cent, the peripheral nerves in 20 per cent and the central nervous system in 8 per cent

The disease used to be thought of as being fatal there may be long periods of remission mistaken for cure It is also likely that abortive forms occur and that if the hypertension due to vascular involvement in the kidney can be throttled early wide dissemination may be averted

Together with Barnum and Dolkart[20] I observed a case of a young woman who received iodine and thiouracil for a suspected hyperthyroidism and who on entrance to St Luke s Hospital exhibited signs of ischemia in fingers and toes peripheral neuritis wrist drop, eosinophilia and marked hypertension with albuminuria and red cells A bilateral splanchnicectomy was done resulting in a complete recovery from all symptoms A nine year follow up revealed no recurrence and a normal blood pressure The histologic picture of a biopsy revealed no acute lesion but a healed endarteritis

Most of the time however, a terminal picture is presented with peripheral and visceral arterial involvement at which time ACTH and cortisone may give symptomatic relief and bring on a temporary remission but will not halt the course of the disease

3 Temporal Arteritis

Following a prodromal period of several days or weeks of fever weakness and anorexia tortuous painful firm and tender temporal arteries develop with a periarteritis around them Sometimes one artery is involved before the other but usually the lesion is bilateral A slight anemia and leucocytosis may be present In the three cases which came to my personal attention the common carotid sheath was tender and the cervical lymph glands were enlarged

Horton, Magath and Brown first described this syndrome [21] Why it should affect elderly persons and why it should affect cranial arteries (cerebral and retinal vessels are often involved) is unclear The term giant-cell arteritis has been suggested by some of the British workers The histologic sections are indistinguishable from periarteritis nodosa, and cerebral Buerger s disease may be confused with it

The disease takes a lengthy though benign course Section of the artery for biopsy promptly relieves the headache but does not alter the course of the disease Eosinophilia and asthma are not as common as in periarteritis nodosa. In one of our own patients febrile for many years the lesion remained quiescent for several years after the temporal segment cooled off but the patient died of a subarachnoid hemorrhage due to a basilar aneurysm

Ocular complications occur in about 40 per cent of the cases [22] The central retinal artery may become occluded or an ischemic optic neuritis develops leading to partial or complete loss of vision in one or both eyes With the exception of this possible complication or that of cerebral vascular involvement the disease runs a benign course even though a protracted one

The chief value of ACTH and cortisone here is to control the symptoms and safeguard any vision that remains Neither anticoagulants, nor vasodilators, nor sympathetic block have given us any help However, in one case small doses of roentgen ray therapy directed toward the cervical lymph glands seemed to hasten recovery. While my experience is exceedingly limited, a lymphatic spread through the teeth, pharynx or tonsils and ascending along the perivascular lymphatics seems to be a distinct possibility

SPECIFIC ARTERITIS

An acute arteritis may be contiguous with an extending area of inflammation such as exists around a tuberculous or luetic focus Arteritides occur during certain infectious diseases, such as influenza, scarlet fever or virus hepatitis We have seen an acute gangrene of all ten toes in a child with meningococcus septicemia (fig 118), and gangrene has also been seen in the wards of Cook County Hospital following scarlet fever Recently such a 4 year old child was seen in the pediatric ward of St Luke's Hospital with a high antistreptolysin titer (fig 119)

The distribution of such ischemia would indicate that massive vasospasm, dehydration and hemoconcentration play parts, and that the lesions are not embolic An acute necrotizing arteritis, not unlike the one seen in a



FIG 118 Bilateral gangrene of the toes following meningococcus septicemia in a 12 year old child The lesion is so symmetrical that massive vasoconstriction, perhaps brought on by the hypotension of toxemia, may have initiated the digital thromboses

Shwartzman phenomenon, can be observed The treatment of these gangrenous lesions is amputation should the patient usually a child survive the acute infection

In the florid cycle of rheumatic fever in erythema nodosum and in typhus fever, such acute necrotizing arteritis may develop [23] Exudative vegetative proliferative and organizing forms are simply reactions of various intensity or stages of repair

Tuberculous arteritis occurs especially in the lungs veins however are more vulnerable than arteries Pulmonary hemorrhage in tuberculosis occurs more frequently from veins although arteries have been seen to rupture or form aneurysms Neel and Herrmann described a tuberculous thrombus in a pulmonary vein from which a peripheral embolism appeared in the lower extremity [24] We have seen a young Negro girl with large cavitation in one lung and occluded digital arteries in the toes Biopsy however was not obtained

Syphilitic arteritis takes several forms In the aorta a syphilitic aortitis develops with or without aortic regurgitation Later an aneurysm may develop which may affect the ascending or transverse aorta Luetic arteritis of the smaller vessels does occur and simulate thromboangiitis or arteriosclerosis It is an occlusive vascular disease and is seldom seen nowadays although vascular clinics could demonstrate them 20 to 30 years ago to



FIG 119 This 4 year old child was brought to the hospital with a fulminating toxemia dehydration, spiking temperatures and a high antistreptolysin titer Toes, fingers and upper and lower lips became gangrenous. (Courtesy of Dr C. Edward Stepan, St. Luke s Hospital, Chicago)

students without much difficulty Raynaud's phenomenon, *i e*, a triphasic response to cold, is very frequent When other signs of congenital or acquired syphilis are present, such as necrosis of the ears or nose or hemoglobinuria, the vascular lesion can be suspected of being of luetic origin Finding of a gumma in the histologic specimen is rare, in most instances an obliterative endarteritis is all one can see under the microscope Of great surgical significance are the *luetic aneurysms*, and they will be discussed in chapter 15, Aneurysms

As pointed out by Louis G. Herrmann, one of the characteristic features of obliterative vascular disease due to syphilis is the spontaneous development of an active collateral circulation Intensive therapy with passive vascular exercises has hastened the establishment of this collateral circulation [25] While our clinic has gradually abandoned the use of suction and pressure therapy for chronic obliterative vascular disease, the method is still being used in some hospitals, notably the Presbyterian Hospital in Chicago [26]

ENDARTERITIS OBLITERANS

From the foregoing paragraphs it is obvious that any mechanical, thermal, chemical, bacterial and allergic insult to the arterial tree will result in a type of chronic obliterating vascular disease which is not atherosclerosis, which is not due to diabetes and in which the original noxious agent is nebulous or unknown The question naturally arises why it is necessary at all to recognize such an entity and what therapeutic attack is most useful if such a dignosis is admitted

The original discussion of this syndrome is found in von Winiwarter's article of 1879, these histories probably contained cases of thromboangiitis obliterans and luetic arteritis Kramer[27] has attempted a differential diagnosis between endarteritis obliterans, thromboangiitis obliterans and arteriosclerosis obliterans From the analysis of Kramer's cases one may surmise that most of his patients suffer from arteriolar disease with or without diabetes and hypertension, that the etiology of the disease is unknown and that the therapy of the disease is mostly conservative, consisting of triple typhoid vaccine and amputation if gangrene is present

We have made such a diagnosis rarely and feel that it is a negative one, arrived at by exclusion of all other factors When a patient's disease is mostly in the digital arteries or arterioles, he may have marked terminal ischemia with maintained major pulses On the upper extremity, dorsal sympathectomy is usually sufficient to rehabilitate the patient On the lower extremity, lumbar sympathectomy combined with metatarsal amputation results in a fast recovery with full economic rehabilitation in a short time.

In the case of Ethan J, a 51 year old accountant, one was unable to elicit any noxious agent to account for a severe digital ischemia with incipient gangrene A lumbar sympathectomy with midmetatarsal amputation relieved him of pain and disability and put him on his feet after weeks of sleepless nights In the arteriogram (fig 120) the posterior tibial and peroneal

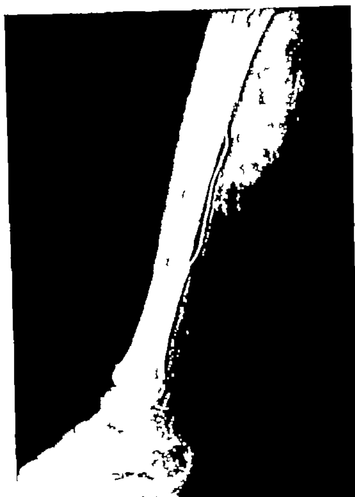


FIG 120 Arteriogram of Ethan J., a 51 year old man. Note segmental closure of the posterior tibial artery between lower third of the leg and the plantar artery The tortuous vessel in the lower third of the calf is a sturdy collateral, perhaps the peroneal artery The contours of the vessels are smooth. The arteries obtained on metatarsal amputation were obliterated with fibrous tissue. There was hyaline thickening of the intimal layer but no atheromatosis

arteries are visualized The posterior tibial artery is quite tortuous possibly the main stem is closed in the lower third of the leg The plantar artery is visualized but a segment at the level of the ankle joint is closed There is no evidence of atheromatosis Thromboangiitis was considered but could not be positively diagnosed The patient has had no difficulty for six years following operation

COLLAGEN DISEASES

Textbooks have been written on this intriguing group of diseases [28] Here only such manifestations are mentioned which may be confused with vascular diseases requiring surgical care The outstanding sign is Raynaud s phenomenon and both scleroderma and lupus erythematosus often exhibit such a phenomenon Many patients throughout the country including some of our own have had sympathectomies performed for Raynaud s syndrome only to develop a full blown diffuse scleroderma years later This is the main reason for discussing this phenomenon here Other entities such as ery

thema nodosum or induratum, may be confused with phlebitis or phlebitic ulcers

1 SCLERODERMA

Since Raynaud's phenomena frequently antedate the typical thickening of the skin, contractures of the fingers, tightness of the face, stricture of the esophagus, pulmonary fibrosis and general debility which belong to the full-blown picture of diffuse scleroderma, it is well for every surgeon contemplating treatment of Raynaud's syndrome to ask himself the following questions

(1) Are the vasospastic phenomena the early symptoms of an incipient diffuse scleroderma? It has been our teaching to watch for doughy edema on the flexor surfaces of the wrists, over the sternum and over the malar bones, to look for pulmonary fibrosis in the roentgen film, for scleroderma heart and for esophageal stenosis

The number of patients subjected to dorsal sympathectomy for Raynaud's disease who later developed diffuse scleroderma must be very large. We have seen it in our own patients and observed it in patients operated on elsewhere The usual course of events is an excellent early result in the hands of the individual suffering from the triphasic vasospasm. As much as 5 to 10 years later typical scleroderma develops which proceeds in exacerbations and remissions just as fast in sympathectomized as in nonsympathectomized areas

In a staff meeting over 20 years ago a rather sarcastic internist presented a case of diffuse scleroderma involving the face, both upper extremities and the skin around the ankles. He suggested that a sympathectomy, done at an Eastern clinic, was the cause of this terminal disability, which of course was untrue Since that time my associates and I have been very careful not to advise operation in a disease which is primarily not vascular but only interferes with circulation through constriction of the terminal vascular bed and progresses independently from any of the surgical procedures advocated in the past.

(2) The second question to be asked is this a localized sclerodactyly, secondary in digits suffering from intermittent or prolonged ischemia? One sees this not only in Raynaud's syndrome, but in thromboangiitis obliterans and in arteriolar and arterial sclerosis of digits This localized sclerosis of the digital skin has nothing in common with diffuse scleroderma, although in some communications extolling the virtue of treatment by iontophoresis this distinction has been ignored [29]

One should recognize then a *diffuse scleroderma*, a collagen disease, characterized by an increase and swelling of collagen fibers, loss or fragmentation of elastica, atrophic rete pegs, deposition of melanin in the basal cells and retention of calcium The last may form large, confluent, palpable masses, which break through the skin at points of pressure and give rise to "chalk gout " Or they can be seen as streamers in the subcutaneous tissue, often barely visible pinpoints of lime, or sometimes huge masses, in the tips

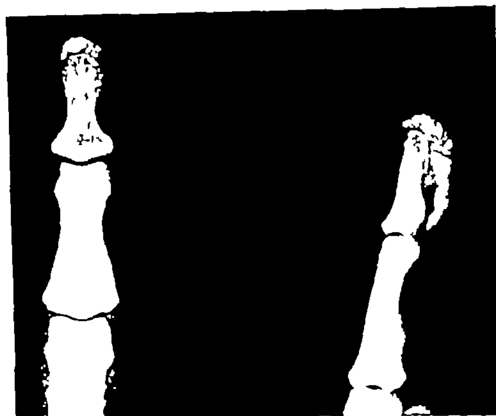


FIG 121 Huge deposition of calcium in a patient suffering from diffuse scleroderma with calcinosis. Note the osteoporosis at the tip of the terminal phalanx.

of a digit or at tendinous insertions (fig. 121) One should distinguish this *diffuse scleroderma* from *sclerodactyly* a secondary phenomenon following chronic ischemia one sees this digital sclerosis after frostbite prolonged vasospasm or any chronic inflammatory or degenerative disease

Nomenclature, such as acrosclerosis and acroscleroderma with Raynaud s phenomenon simply confuses the picture more and will not be used in this monograph. The idea that acrosclerosis that is involvement of fingers and toes with Raynaud s phenomenon can never become a diffuse visceral disease is not borne out by our experience

Etiology

Diffuse scleroderma is not a vascular disease and hence will not improve on measures directed toward improving circulation Since it is often accompanied by microscopic and visible accumulations of calcium under the skin or in any connective tissue a disturbance of calcium metabolism has long been suspected In unpublished studies with Professor C I. Reed of the Department of Physiology of the University of Illinois College of Medicine calcium determinations were made in biopsied skin segments in which the calcium content was roughly twenty times greater than that obtained from normal skin of the same age group In one case, a calcium balance study of a patient suffering from diffuse scleroderma was found to be negative, indicating increased calcium excretion When, therefore Garlock reported his en

couraging results with parathyroidectomy,[30] two severe cases of diffuse scleroderma were subjected to this procedure The three removed glands of each patient were of normal size, showed no hyperplasia and there was no clinical improvement Since *parathyroidectomy* for scleroderma has been practiced on a considerable scale by Leriche and his co-workers since the early 1930's, it is of interest to review his last report on the subject, based on 57 operations [31] Leriche advised against parathyroidectomy in the rapidly progressing cases and most often combined this operation with various types of sympathectomy While a few late remissions are described, one cannot feel that Leriche has proved his thesis of hyperparathyroidism existing in diffuse scleroderma or that parathyroidectomy was worth while

From observation of patients in a very early stage of the disease, one cannot escape the impression that an inflammatory reaction is present in connective tissue and that calcification is a terminal phase of the disease which may result in compensatory parathyroid activity Under the heading of treatment, the attempts of treatment based on such reasoning will be described

Another line of reasoning connects all calcified tissue with replacement of ischemic necrosis and believes in more extensive sympathectomies than have been done in the past [32] While it is true, as emphasized before, that vasomotor phenomena precede the sclerodermatous changes—often by years, there is no evidence that the Raynaud's phenomena are the cause of scleroderma

Loss of elastic fibers followed by replacement with calcium deposits is another line of thought especially applied to the biochemistry of arteriosclerosis [33] Since the elastic tissue breaks down as a result of enzyme action of elastase, one wonders whether this same process might not be operating throughout the connective tissue Balo and Banga[34] have shown that a source of elastase is in the pancreas and they have indicated that the elastase content of the human pancreas in arteriosclerotic patients is substantially decreased. The reason this thought is so intriguing is that a noted dermatologist, J Sellei of Budapest, has long advocated the use of pancreatic extract in cases of scleroderma, probably with the mistaken idea of supplying tryptic action [35] It is known, however, that elastase is not contained in pancreatic juice and that it probably comes from the alpha cells of the islets This line of thought will require much further study

Pathology

Experimentally, Hans Selye developed sclerodermatous lesions with calcific deposits in 20 per cent of his rats with as little as five units of parathyroid extract given intraperitoneally over a period of two weeks However, as mentioned above, the therapeutic implications of these experiments have not borne fruit and parathyroidectomy is definitely of no help in scleroderma

The pathology of "progressive systemic sclerosis," a name applied to scleroderma by Robert H Goetz,[36] involves any area where connective tissue

is present and thus is only partially studied and treated by dermatologists It consists essentially of a sclerosis which may occur in skin, muscles or blood vessels Initial edema is followed by proliferation and later contraction of collagen fibers resulting in atrophy of the organs involved This may be in the skin, in the interstitial tissue of the lungs as pulmonary fibrosis, in the lower end of the esophagus as esophageal stricture or in the small intestine causing duodenal and ileojejunal stasis It produces colonic stricture, nephrosclerosis, cirrhosis of the liver spleen and endocrine glands and myocardial fibrosis. Involvement of the central nervous system has been reported

Of course not every patient shows such a diffuse localization and it is very true that the initial lesions are in the digits of the extremities more often in the fingers

The ground substance, a mucopolysaccharide, seems to swell proliferate undergo fibrinoid necrosis and calcium deposits What initiates this process is unknown and therefore no specific treatment is available

The Clinical Course

Because of its insidious onset—often with vasomotor phenomena—one cannot be cautious enough about suspecting the presence of scleroderma when patients with joint aches swelling of the hand and triphasic color changes come under observation Rheumatoid arthritis is frequently diagnosed and both salicylates and cortisone may bring on a remission Nevertheless the disease progresses inexorably causing increasing stiffness in the fingers tightening and pigmentation of the skin and early difficulty in swallowing. At this stage the barium swallow may not show organic obstruction but only spasm. The patient s food intake is gradually reduced, the fingertips may break down and can exude chalklike material (chalk gout) The face first swells then later tightens and opening of the mouth may become restricted The cardiogram may not change but the patient shows exhaustion and dyspnea, and the myocardial reserve is diminished At pressure points such as over the knees and elbows or on bunions the skin breaks down and calcium deposits are laid bare The patient becomes more and more adynamic

It is difficult to foretell the rate of progress of the disease With the help of general supportive measures which combat the anemia with iron, the nutritional defect with frequent small feedings and vitamins, and the sclerosis of the thyroid and adrenal with thyroid extract and cortisone one may effect a stationary status for several years Sometimes there is a spectacular regression of symptoms particularly in the localized form of sclerodactyly In one woman from Harbor Point Michigan the sclerosis actually softened up and disappeared She drank a pint of goats milk a day at her neighbor s suggestion We had no courage to stop her since we knew of nothing better

Most patients however, will slowly progress and die either of a *pneumonia* superimposed on their pulmonary fibrosis or of uremia as a result of

increasing nephrosclerosis Many patients have had sympathectomies, a few of them have had parathyroidectomies, but the course of the disease remains unaltered

Differential Diagnosis

The full-blown picture of scleroderma is unmistakable However, the early manifestations of localized edema, arthritis and Raynaud's phenomena are shared by rheumatoid arthritis, by lupus erythematosus and dermatomyositis Jager[37] has given a useful diagnostic table, which, like all such tables, gives general guiding principles There is, however, an obvious overlap and some authors feel that scleroderma and dermatomyositis are simply different facets of progressive systemic sclerosis (Table III)

Table III*

COMPARISON OF CLINICAL CHARACTERISTICS OF POLYARTERITIS NODOSA, DISSEMINATED LUPUS ERYTHEMATOSUS, SCLERODERMA AND DERMATOMYOSITIS

CLINICAL CHARACTERISTICS	POLY-ARTERITIS *NODOSA*	DISSEMINATED LUPUS ERYTHE-*MATOSUS*	SCLERO-DERMA	DERMATO-MYOSITIS
Sex incidence M F	4 1	1 3 5	1 1	1 1
Commonly noted age of onset (in years)	20–40	15–40	30–50	10–50
Arthralgia or arthritis (relative frequency)†	++	++	+	±
Cutaneous lesions	++	+++	+++	++
Cardiac involvement	++	++	±	+
Hypertension	++	±	–	–
Pulmonary lesions	+	+	±	–
Pleuritis with or without effusion	±	++	–	–
Abdominal pain	++	+	–	–
Impaired renal function	++	+	–	–
Lymph node enlargement	+	++	±	±
Splenomegaly	±	+	–	–
Peripheral neuritis	++	±	±	±
Focal brain lesions	+	±	–	–
Muscle inflammation	+	±	±	+++
Abnormal laboratory findings				
Anemia	+	++	±	±
Leucocyte count (usual)	Elevated	Reduced	Normal	Normal
Eosinophilia	+	±	±	+
Abnormal urinary sediment	++	+++	±	±

† Frequency roughly estimated as follows

+++ = 60–100%
++ = 40–59%
+ = 20–39%
± = less than 20%
– = a frequency no greater than in the ordinary population of the same age

* Modified from Table 112, p 1225 in Harrison, T R Principles of Internal Medicine Second Edition, The Blakiston Co , New York and Toronto, 1954 Chapter by Jager, B V Collagen Diseases

Raynaud s phenomena, arthralgia, erythema and purpuric patches may occur in all four diseases Visceral lesions are common At times any one of these collagen diseases may resemble rheumatic fever or rheumatoid arthritis From a surgical standpoint it is most important to differentiate true Raynaud s disease from an early acrosclerosis with Raynaud s phenomena In fact, in our vascular clinic we see typical cases of scleroderma in patients who 5 to 10 years ago had no skin manifestations even though these were looked for In true Raynaud s disease acrosclerosis may develop always limited to the site of prolonged vasospasm of many years duration In Raynaud s disease, the heart, lung, esophagus and kidney are not involved

Treatment

Since the course of this disease is unknown, treatment is mostly symptomatic Daily massage of and heat to the stiff swollen hands keeps them flexible Anemia hypometabolism and poor nutrition can be at least partially corrected

When vasospasm is conspicuous and especially if the lesion has been mistaken for Raynaud s disease, sympathetic ganglionectomy has been performed In true scleroderma this operation mostly undertaken for the hands may be followed by healing of superficial ulcers and warming of the skin One *never* sees however any beneficial effect on the progressive sclerosis of connective tissue for this reason sympathectomy for diffuse scleroderma is regarded as a contraindication Again as will be pointed out in the discussion of Raynaud s disease this does not hold for the localized fibrosis of ischemic fingers, which may be due to many causes of ischemia, such as frostbite thromboangiitis obliterans, Raynaud s disease and arteriosclerosis

Experience with partial parathyroidectomy in two cases has been equally disappointing and there seems to be no favorable report since the original communications of Leriche[31] and Garlock [30]

Since the disease baffles dermatologists and internists alike and since our follow up material consists of a few patients on whom sympathectomies have been performed in the past, some experience has been gained, with para aminobenzoic acid (PABA) widely heralded but useless in our hands Large doses of typhoid vaccine have given us an occasional short remission With the cooperation of Dr Ralph Dolkart, then of the Department of Medicine at St Luke s Hospital, Chicago a number of patients have been studied under the effect of ACTH and cortisone Prompt pain relief and some remission however were followed by a recurrence a necessity for increasing dosage and intolerance In a young nurse huge and uncontrolled dosage of cortisone administered in a neighboring city on an outpatient basis resulted in multiple gangrenous ulcerations of fingers and toes and an exhaustion of the clotting mechanism[38] (fig. 122)

Butazolidin, like cortisone gives early pain relief but has many side reactions and does not seem to alter the course of the disease.

Recently mobilization of calcium deposits by a chelating agent *ethy*



FIG 122 Gangrene of fingers and toes in a young woman suffering from scleroderma, and treated by uncontrolled doses of cortisone The painful patches of gangrene appeared shortly after cortisone was started

lenediaminetetraacetic acid (EDTA), has been attempted and a case of scleroderma with calcinosis has been so treated with surprising results [39] The drug combines with metal ions to form complexes which are water soluble and virtually undissociated The sodium salt, marketed as Versenate calcium disodium, is nephrotoxic and causes tubular damage but may be used with certain precautions 50 milligrams per kilogram per day given for five consecutive days and followed by two days of rest before a second course, has been the suggested safe dose [40] So far, three patients on our service have had one or two series of treatments, with increased urinary excretion of calcium and some temporary improvement

2 Lupus Erythematosus

The second collagen disease which is accompanied by Raynaud's phenomena in about 20 per cent of the cases is lupus erythematosus While this disease does involve the subendothelial layers of arteries and veins and produces arteritis with periarterial edema and fibrosis, it may affect all connective tissue throughout the body and has again some earmarks of allergic inflammation, the collagen reacting abnormally to a variety of antigens

The widely scattered vascular lesions, lesions on the serous membranes, a peculiar type of glomerular nephritis, and the endocarditis of Libman-Sacks represent the complete clinical picture Clinically there is evidence of fever, lassitude and prostration, without evidence of bacterial infection The white blood count and platelet count are often low and a typical L E cell in

the bone marrow is characteristic, but not always present. These cells are polymorphonuclear leucocytes containing digested nuclear debris the L E cell has been recently seen to appear after administration of Apresoline hydrochloride for hypertension and in penicillin reactions and is thus evidence of toxicity or hypersensitivity

The interest of the surgeon lies in the fact that Raynaud s phenomena are sometimes the early dominant symptoms of lupus erythematosus We have recently observed such a patient Acute thrombosis of larger arteries and veins does occur The cutaneous lesions such as the butterfly eruption of the face and the purplish discoloration of the fingers, are often absent The skin lesions vary from bluish to red edematous plaques which may break down and form ulcerations In a 16 year old girl who presented a chronic bluish plaque on the supramalleolar area a chronic, hypergranulating ulcer developed which resisted all treatment and which finally ended in an acute systemic lupus erythematosus with high fever glomerulonephritis bronchopneumonia and death (fig 123)

While the localized discoid type of lupus may persist for several years *sudden dissemination may occur following minor injury frostbite* burns, operations or drug therapy There is really no effective treatment although Atabrine *chloroquine* ACTH and *cortisone may bring on a remission* Most patients ultimately die of the disease or some intercurrent infection Recently Dubois has reported favorably on the use of large doses of steroids.[41]



FIG 123 Intractable purplish, hypergranulating ulcer over the right inner malleolus of B T., a 16 year old girl. The ulcer showed no healing tendency over a period of two years and its origin was undetermined. Following exposure to sunlight a disseminated lupus erythematosus developed, with death resulting within two months.

3. Erythema Nodosum

Cutaneous and subcutaneous painful, pale red nodules occur on the extremities with slight fever and a chronic, remitting course The biopsy shows arteritis and phlebitis in the smaller vessels and perivascular collection of neutrophilic leucocytes and lymphocytes

The disease is usually bilateral and symmetrical, appearing most often on the legs of younger women between the ages of 10 to 30 While erythema nodosum may occur in association with a number of infectious diseases, tuberculosis has been often suspected A localized sensitivity to a streptococcus, however, seems the most common cause and the disease is found in rheumatic fever and rheumatoid arthritis Drug sensitivity may cause it

The disease must be distinguished from superficial phlebitis, and it is with this diagnosis that one sees erythema nodosum referred to a vascular clinic The lesions are *round* and pea-sized, and show no relation to visible veins. They do not suppurate.

Large doses of salicylates, elastic bandaging and much rest is usually prescribed The disease seems to run its own sweet course and usually terminates in four to six weeks. It is my impression, although based on very limited experience, that anemic, pregnant women are often afflicted They are relieved to know that no phlebitis is present

4 Erythema Induratum

These are chronic nodular lesions, mostly occurring on the legs, which readily ulcerate in contradistinction to erythema nodosum. Again, the ones we have seen are referred to us as chronic, recurrent, nonhealing ulcers and, should a few veins be accidentally present, the label of varicose or phlebitic ulcer is readily attached The lesions we have seen have always been associated with tuberculosis elsewhere in the body and lymphadenitis is often present Guinea pig inoculations, however, are not always positive

Histologically there are perivascular collars of lymphocytes, plasma cells, and much endothelial and fibroplastic proliferation Typical tubercles with giant cells may be present Medial necrosis of small arteries signifies again some type of sensitivity reaction

Clinically, the deep seated, purplish or bluish nodules occur in groups, with a tendency to confluence Healing takes place in the center and the margin becomes active Massive necrosis may take place which heals very slowly.

The ulceration is surrounded by a firm, indurated red border Curiously, the lesion appears on the posterior surface of the calf quite frequently (fig 124) When healing takes place, much subcutaneous tissue is lost and the skin is atrophic and pigmented Because veins and lymphatics are compressed or obliterated by the fibrosing process, swelling of the ankle or lower leg is common

Treatment consists of adequate elastic support, which of course is neces-

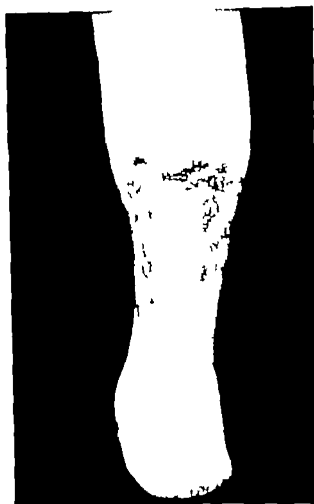


FIG 124 Erythema induratum (Bazin) in Y B., a 48 year old woman. The lesion kept spreading, healing in the center and flaring up at the periphery Healing took place with much loss of fat, atrophy of the skin and pigmentation. A biopsy of the skin was positive for tuber culosis.

sary in any lesion on the lower extremities should the patient remain ambula tory The tuberculous lymphadenitis is treated by moderate doses of roentgen ray therapy Locally our dermatologic consultants have advocated one-fourth per cent silver nitrate and in the case shown in figure 124 this treatment re sulted in healing after many other topical applications have failed Whether chemotherapy for tuberculosis notably with streptomycin helps is not known from personal experience

5 Relapsing Febrile Panniculitis

In a tall 40 year old woman who bred Doberman pinschers in Ohio large patches of subcutaneous fat necrosis were observed on both thighs with many exacerbations and remissions, healing with a depressed pigmented scar There was a low grade fever and leucocytosis and the woman had been treated with hot packs, bedrest and penicillin for several years The diagnosis was migrating phlebitis When she was placed on ACTH and cortisone at our suggestion the lesion cleared up rapidly and has not recurred. A recent



FIG 125 Chronic relapsing panniculitis A huge, hard, red patch of induration, associated with chills and fever, is seen over the anterior surface of the right leg Previous attacks were observed on the thighs, leaving whitish depressed scars (Courtesy of Dr A Barnes, Columbus, Ohio)

Christmas card showed her to be in good health and enjoying life (fig 125)

The plaques are hard, red and about 10 cm in diameter and each new crop is associated with chills or high fever The attack subsides in about three weeks, leaves a depressed scar which looks like a localized scleroderma. Histologically, again one sees a hyperergic inflammation, perivasculitis, perivascular edema and later fibrosis Whether this is a bacterial or viral infection is unknown, but bacterial or viral sensitivity is most likely.

Other forms of local and disseminated arteritides, such as nodular vasculitis, "disseminated arteritis," have been described in an excellent chapter of the Allen, Barker and Hines monograph [18] There is much overlap here, but until the syndromes are better characterized it is well to regard them as being due to allergic vascular disease

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CHAPTER 10

DEGENERATIVE LESIONS (ARTERIOSCLEROSIS AND DIABETES)

DEFINITION

THE OVERWHELMING MAJORITY OF PATIENTS SEEN WITH STENOTIC OCCLUSIVE or aneurysmal lesions of the peripheral vascular tree are suffering from arteriosclerosis In recent years, the term atherosclerosis has been more com monly used to emphasize the importance of fatty atheromatous deposits and to point out that this vascular lesion is not necessarily the result of aging, the fatalistic view of Moschcowitz [1] He defined arteriosclerosis as a progressive and irreversible lesion of the arteries in which hyperplasia of one or more of the structural elements is a primary reaction with deposition of lipids collagenous tissue hyalin and calcium as a secondary response the summa tion of both components result in thickening, dilatation deformity and loss of elasticity of the vessel wall

Moschcowitz felt that arteriosclerosis was the product of intravascular pressure and time and thus he is the forerunner of the thought that imbibition or plasma filtration in the vessel wall is an important consideration Medial sclerosis of the Mönckeberg type was felt to be a distinct lesion with distinct clinical characteristics, which is certainly true in our experience

PATHOPHYSIOLOGY

While it would be impossible to review even cursorily the tremendous amount of work done in the last few years on the experimental production and the clinical manifestations of atherosclerosis as a working hypothesis and a unifying concept, the one of Irvine Page[2] is most acceptable since it has the merit of knitting together a number of disparate facts and corresponds to some of the ideas expressed on the permeability of the vessel wall The basic part of the concept is that atherogenesis is due to an accumulation of substances filtered by the lateral pressure from plasma through the intima. Depending on the nature of these substances retained in the vessel wall and the responsiveness of the tissues to them, a reaction may be set up In this view atherogenesis depends on the following factors

(1) The anatomy, biochemistry and physiology of the vessel wall, all of which are hereditarily conditioned

(2) The composition of the plasma filtrate

(3) The height of the lateral pressure and the amount filtered

(4) The metabolic capacity of the vessel wall

(5) The responsiveness of intimal tissues to filtered products and their metabolities

(6) Changes in the ability of the vessel wall to transport filtered substances such as might result from age, hypertensive disease and metabolic disorders

Since the surgeon cannot alter the heredity of the patient, he can try to influence the composition of the plasma filtrate and see that his patient gets the best possible advice on holding the cholesterol level at or under 250 mg per cent, keeping diabetes and gout under the best possible control and holding the hypertension to a level consistent with the optimal pressure of the patient (see chapter 19, Hypertension) The most extensive sympathectomy and the most skillfully placed arterial graft will be only a short palliative procedure, if the patient continues to deposit atheromata in and around the graft and if the collateral circulation developed to take over the function of the closed arterial pathway will be stopped up by extension of plaques and thrombi

PATHOLOGY

It would not be fitting to enter this difficult field in any detail, and only findings of surgical importance will be summarized The lesions manifest themselves as thickened areas of intima, yellow-gray patches of atheroma, ulceration and hemorrhages of the plaques and occluding thrombi The significant postmortem studies of Rodda[3] have been made on lower limbs free from gangrene and no clinical evidence of ischemia He found a 40 per cent incidence and multiplicity of arterial occlusions in nonischemic limbs of individuals over 60 years of age The lesions comprised atherosclerosis without thromboses or intimal hemorrhage, collagenous sclerosis, granulation tissue, thrombosis secondary to intimal hemorrhage, massive intimal hemorrhage and thrombosis or embolism The greatest incidence of occlusion was in the lower posterior tibial and lateral plantar arteries, but the incidence of intimal thickening was greatest throughout the length of the popliteal artery and in the upper part of the posterior tibial artery

The sites and incidence of arterial obstruction in atherosclerotic ischemia have been most intensively studied by Professor Dible[4] who established various collateral patterns He coined the term "peroneal" leg, this artery remaining patent and feeding the vascular arcades of the foot when the anterior and posterior tibial arteries are occluded, this corresponds to the profunda femoris on the thigh which one often sees greatly enlarged when the superficial femoral artery is blocked.

As was pointed out in chapter 8 Arterial Injuries the vessel wall reacts with great uniformity of response to any type of injury be it a physical or chemical agent, a bacterial infection or a hypersensitive state The variations that do occur may result from differences in the intensity and duration of the stimulus

The lipid infiltration and the formation of atheromata may well be the immediate cause of stenotic or occlusive atherosclerotic lesions There has been tremendous activity both experimentally and clinically to put the faulty lipid metabolism in the center of etiologic factors Nevertheless, the primary injury to the vessel wall the wall s change in permeability and the pressure factor facilitating filtration are all operative There seems to be some evidence that hypercholesteremia inhibits normal repair and we have certainly emphasized the low fat diet not exceeding 90 Gm of fat to all our postoperative patients for many years

It is generally assumed that the diabetic vascular lesion is atherosclerosis appearing earlier and with more intensity Since there is a specific retinal and renal lesion in diabetes the suggestion of Lundbaek[5] of a specific diabetic angiopathy seems reasonable Among the clinical forms, a description of a diabetic vascular lesion will be given which affects the periphery and differs from hypertensive arteriolar sclerosis

RATIONALE OF SURGICAL MANAGEMENT

In spite of the early claims in the field of restorative arterial surgery that atherosclerosis is a segmental disease and that by attacking the single closed segment the patient is fully rehabilitated, it is becoming increasingly obvious that prevention of further progression of the disease is of paramount importance Here dietary and pharmacological measures will play a great part in the picture

This is not to say however that the release or bypass of an occluded segment is not an important step in halting the progress of the disease since the fall in pressure and decrease in flow velocity of a vessel distal to the obstruction definitely increases the hazard of thrombosis and may promote endothelial proliferation, as in the ligature disease of Leriche [6]

It is however imperative that the vascular tree be carefully studied regarding regional vascular involvement (see part II Methods of Diagnosis) and that because of a lost femoral pulse in one inguinal region the replacement of the closed iliac segment be not hastily undertaken just in order to increase the series of plastic grafts studied at the moment While it is very true that clinically manifest atheromata do occur at bifurcations such as at the aortic iliac femoral and popliteal segments, it is good to remember that (1) the disease almost invariably is bilateral and often more surgical help can be given the good leg than the involved one that (2) segmental occlusions are multiple even in the involved leg, and the bypassing of an occluded segment in Hunter s canal will fail unless the iliac stenosis is relieved and (3) if cardiac, cerebral or renal damage is pronounced the surgical effort had

better be expended on prevention or elimination of gangrene, rather than on direct restoration of continuity.

CLINICAL FORMS

Unilateral Iliac Stenosis

One can occasionally encounter such a lesion (fig 126) after a blunt trauma, such as falling several feet from a ladder, or from a poorly applied arterial clamp on the iliac artery, nevertheless, the lesion is most commonly due to a partially obstructing atheroma which creates minimal symptoms until over one half of the lumen is obstructed [7] There may be a low backache or fatigue in the ipsilateral buttock or posterior surface of the thigh, but no claudication in the calf As will be pointed out under treatment (p 231), such a patient is eminently suitable for a localized endarterectomy, at which time a saddle plaque which slightly obstructs the opposite side, can be gently lifted out High lumbar sympathectomy preceding or concomitant with this procedure is the method of choice There should be no advanced stenosis or occlusion in the femoropopliteal region



FIG 126 The lower end of the aorta shows some narrowing and atheromatous indentations The flow is adequate and the right common iliac artery has a good contour and good opacity The left common iliac is badly deformed, the hypogastric artery looks poor There was unilateral backache in this patient, related to exercise The left femoral pulse was weaker than the right There was no claudication in the calf

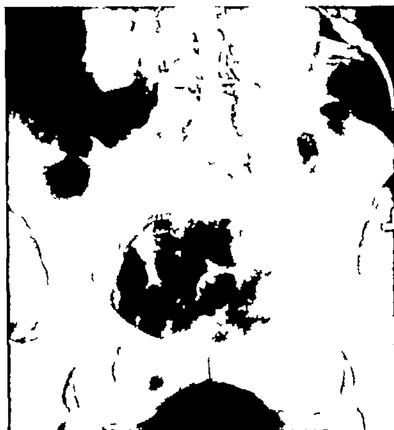


FIG 127 There are patchy areas of decreased opacity in both common iliac arteries but there is no complete occlusion A bypass procedure here simply serves as additional potent collateral

Bilateral Iliac Stenosis

Such a patient (fig. 127) shows identical symptoms with the first form except that both sides are affected both femorals are weak and therefore comparison between them is not so easy As in all other locations the lesion seen on surgical exposure is always far more advanced than the arteriogram would let one suspect. While this patient was scheduled for a lower aortic and bilateral iliac endarterectomy the lesion seen on laparotomy called for a bypass between the aorta and upper end of the femoral arteries Right lumbar sympathectomy had been previously done (Cushing clips)

The conical narrowing of the aorta and the rigid, ribbon like iliacs with mottling leave no doubt that a complete aortic occlusion is imminent in this 47 year old man who complained of *calf pain* on walking with palpable pulses in the foot and good oscillation (fig. 128)

Unilateral Iliac Occlusion

The rest of the arterial tree looks so normal in this case (fig. 129) that trauma or embolism must be suspected. Actually this is the case of a 53 year old woman with rheumatic mitral stenosis auricular fibrillation and an embolus to the right iliac artery A lumbar sympathectomy extraction of the

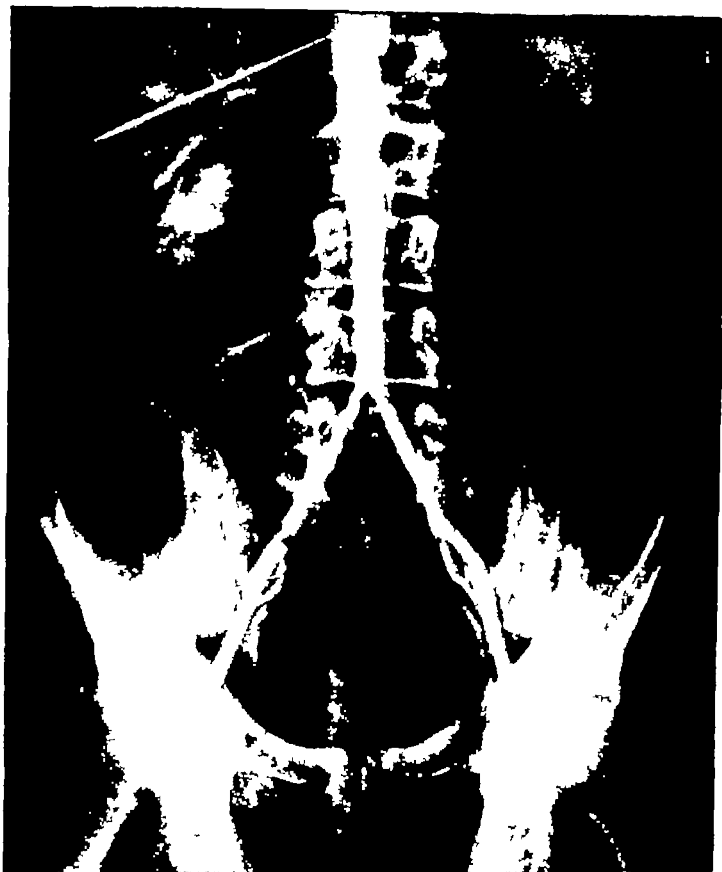


FIG 128 Note the conical narrowing of the abdominal aorta, starting shortly below the origin of the renal arteries, and the ragged contour of the iliacs The femoral arteries look adequate



FIG 129



FIG 130

(See facing page for legends)

clot and later a mitral commissurotomy completely rehabilitated this patient The aortogram in figure 130 in contrast shows a mottling at the bifurcation corresponding to a plaque at this level a somewhat ragged contour of the right common iliac artery and only a faint shadow of the left external iliac artery below the origin of the hypogastric This patient had a successful endarterectomy which restored his left femoral pulse for four years, but he more recently developed a low femoropopliteal occlusion which could not be surgically reopened Thanks to a preliminary lumbar sympathectomy done before the iliac endarterectomy, he can still walk a block and has no trophic changes in the foot

Bilateral Iliac Occlusion with Saddle Plaque at the Bifurcation

It is a strange and rather disturbing fact that younger individuals in their late forties and early fifties develop isolated short segmental occlusions which seem to leave rather uninvolved segments above and below them (fig



FIG 131 Total occlusion at the bifurcation with some disease both above and below the block, but involving comparatively short segments. The patient refused further surgery after bilateral lumbar sympathectomies, since his claudication improved. Such patients re quire a high sympathectomy including the first lumbar segment or even higher

FIG 129 Complete absence of filling of the right iliac and femoral segment, three weeks after an iliac embolus from a fibrillating heart

FIG 130 Total left and incomplete right iliac occlusion in a 47 year old man whose femorals pulsate well four years after endarterectomy A left femoropopliteal occlusion, oc curring three years after endarterectomy emphasized the importance of looking for stenoses below obvious occlusions.

131) The explanation probably lies in the early development of adequate collateral circulation which fails to develop in the presence of long, stenotic segments Roentgenologically this patient (fig 131) has a good aorta above the second lumbar vertebra and good external iliac arteries There is abundant collateral circulation in the lumbar arteries Bilateral lumbar sympathectomies gave him within six months a marked relief from claudication He would not consider any further procedure and it was not urged

Total Occlusion of the Abdominal Aorta

Such patients (fig 132) in whom there is much back pain, pain in the buttocks and posterior thigh on walking, may well show a progression of the



FIG 132 An aortic bypass from below the renal to the external iliac arteries was performed two years ago on this 43 year old woman, who had absent femoral pulses and a great deal of backache Vigorous pulses are maintained in both femoral segments

lesion toward the renal arteries, at which time hypertension can develop Whether endarterectomy or transection with bypass to the iliacs is preferable depends on the operative findings Bilateral high lumbar sympathectomy including the lower three dorsal ganglia is always performed, although aorta to iliac or femoral bypasses have been very successful as indicated by Michael DeBakey's unprecedented experience [8] In such total occlusions, as in the partial ones, it is most important to ascertain the state of the femoropopliteal

segments Results after several years are still excellent if the aortic lesion is an isolated one A low occlusion at or below the popliteal level often vitiates the results

Segmental Stenosis of the Femoral Artery

In doing routine femoral arteriograms during lumbar sympathectomies for obliterating arteriosclerosis one may encounter a ragged superficial



FIG 133 FIG 134

FIG 133 Note a partially occluding atheroma in Hunter's canal This was in the "good" leg of the patient, who had bilateral lumbar sympathectomies and left femoral endarterectomy in 1954 Four years later he appeared with a closed lower femoral segment on the right and a sudden decrease in walking ability What should have been done in 1954? Today I would unroof Hunter's canal

FIG 134 Severe, widespread involvement of the entire superficial femoral artery with potent collaterals in the profunda femoris. There is subtotal obstruction between the two arrows at the femoropopliteal level. No grafting procedure or endarterectomy has much chance for success.

femoral artery causing no symptoms and remaining symptomless for several years (fig 133) This patient s pulses and oscillometric curves were satisfac tory and only the fact that the oscillations diminished after exercise instead of increasing pointed to arterial stenosis Four years later this segment was occluded

Diffuse Stenosis and Destruction of the Superficial Femoral Artery

Prognosis in such a case is much worse (fig 134) The vascular tree is bound to be severely diseased below the knee In order to make sure that the distal run-off in the leg is insufficient to permit any grafting procedure, an arteriogram can be done after exposure of the femoropopliteal segment Such a film, however, usually denotes diffuse vascular damage below the popliteal level, and sympathectomy should be done unless there is a paradoxical drop in skin temperature after the release of vasomotor tone (see part II, Methods of Diagnosis)

Segmental Occlusion of the Superficial Femoral Artery

Typically, this is at the upper end of Hunter's canal, causing a moderate amount of claudication, contrarily, pulses at the ankle may be present, though diminished, and the oscillometer shows a slow, retarded pulse wave The fact that such a retarded pulse is present is a good indication that no low posterior tibial occlusion exists and encourages one to advise a bypass In such a patient (fig 135), lumbar sympathectomy followed by a bypass sufficiently high and low to avoid the visible stenosing atheromata is very satis-



FIG 135 Segmental occlusion at the upper end of Hunter's canal with some atheromatous stenoses above and below the occlusion This is a good case for a bypass, unless there is evidence of widespread atheromatosis below the knee

factory The material preferred and the technique used will be discussed in part IV

Stenosis and Occlusion of the Internal Carotid Artery

Incomplete or complete occlusion of the internal carotid artery at or just distal to the carotid bifurcation is receiving more and more attention, and while the lesion may also involve the opposite carotid or the vertebral artery restoration of blood flow is possible and should be done as soon as the diagnosis of an incomplete occlusion is made Charles Rob[9] has presented an unprecedented material of 27 patients and while circulation could be restored in all of the 11 incomplete occlusions in only 4 of the 16 complete occlusions could blood flow be restored While palpation of the carotid artery is unreliable carotid angiography has been of great help and our neurosurgical colleague, Dr Oscar Sugar has supplied me with an excellent example (fig. 136) The commonest symptom is unilateral muscle weakness associated with speech disturbance if the dominant hemisphere is affected Headache transient blindness and often a gradually increasing hemiplegia occur Thromboendarterectomy is the procedure of choice but localized resection and end to end anastomosis have also been done [9]

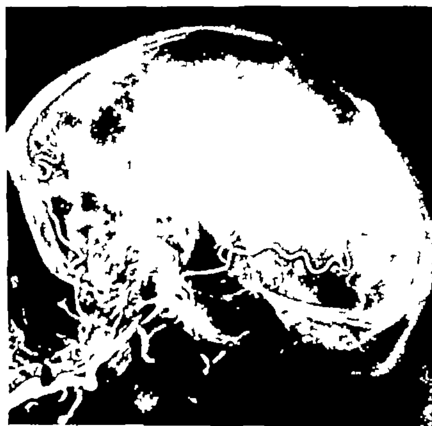


FIG 136 Carotid arteriogram in Geo V a 49 year old arteriosclerotic patient (Ill Res. Hosp 2961/1948), who had intermittent attacks of visual disturbance, dizziness and fleeting paralysis. Note the excellent filling of the common and external carotid arteries, and the branches of the latter There is a total occlusion of the internal carotid artery at the carotid bifurcation. (Courtesy of Dr Oscar Sugar U of Illinois College of Medicine.)



FIG 137 FIG 138

FIG 137 A rather ragged contour of the superficial femoral artery, with total occlusion at the edge of the adductor fascia The distal stump of the femoral artery has not filled but this is often due to lack of serial films If the venous filling time is over 20 seconds, it is wise to delay a single exposure to five seconds after the injection is stopped A second injection is never done

FIG 138 Arteriogram of the same patient taken during exposure of the femoropopliteal segment The lower end of the femoral artery is patent There is an almost completely obstructing atheroma at the popliteal bifurcation The lower leg has only one fair vessel, the posterior tibial Endarterectomy of the popliteal bifurcation may occasionally be successful, if it fails, the leg is lost

Multiple Segmental Occlusions

Multiple segmental occlusions are so frequent that it is important to obtain suitable arteriograms of the femoropopliteal segments, even although a high iliac occlusion is obvious When an iliac occlusion manifests itself in early claudication of the calf, a lower femoral occlusion may be suspected (fig 137) In this film, no distal filling of the popliteal artery and its branches is visible and the femoral artery was pulseless, though patent Exposure of the femoropopliteal segment, however, revealed a patent vessel, and a second arteriogram (fig 138) shows a narrow anterior tibial and a thin tortuous posterior tibial artery. There is a huge almost totally occluding atheroma at the popliteal bifurcation, which would make a bypass to the popliteal artery quite hazardous

Occlusions in the Lower Leg and Foot

These are often asymptomatic until a higher occlusion or a closure of a collateral artery supervenes (fig. 139) This patient s popliteal artery looks very adequate He has no visible anterior tibial artery but his peroneal artery seems to conduct well and it is feeding a narrow dorsalis pedis artery This patient had no symptoms until an occlusion supervened in Hunter's canal He responded well to a femoral bypass losing his claudication

Arteriograms, however seldom reveal a situation emphasized recently by E. Szilagyi A soft, comparatively normal lower femoral segment is found on exploration and an end to side anastomosis is readily inserted Below the bypass however more advanced atheromatosis is encountered just above or at the popliteal bifurcation (fig. 140) This finding frequently militates against a successful patent bypass and calls for a systematic exploration of the popliteal and tibioperoneal segments

As pointed out by Dible[4] and his pupil Rodda,[3] the incidence of multiple occlusions in the three arteries of the lower leg is high even in asymptomatic patients More and more lower leg arteriograms will have to be done as advocated by Cannon and his associates [10] Since one will hardly ever be able to obtain pedal arteriograms of the clarity seen in cadavers (fig. 141)

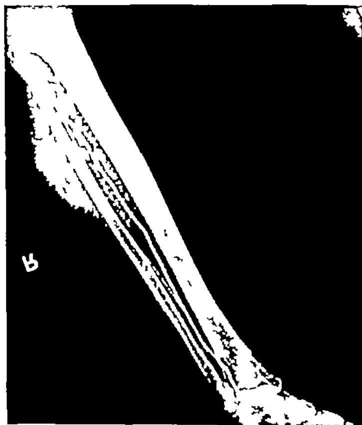


FIG 139 Operative angiogram, done during exposure of Hunter's canal. The lower femoral segment is quite adequate. No anterior tibial artery is visualized and the posterior tibial artery thins out at midcalf. The sturdy vessel is the peroneal re-entering the posterior tibial artery and feeding the dorsalis pedis.

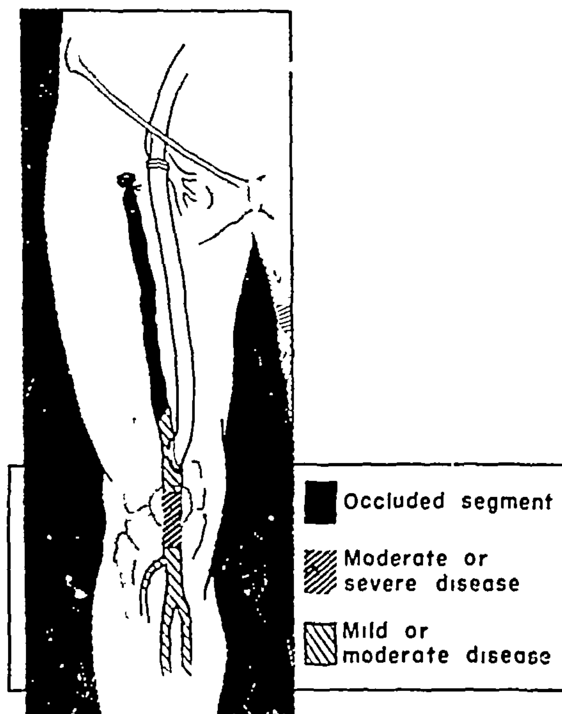


FIG 140 Diagram of a frequent cause of failure in cases of femoral bypass The proximal anastomosis is end to end, the distal one has been placed into a "soft" area at the lower end of Hunter's canal, showing mild or moderate atheromatosis Below it, however, just above the popliteal bifurcation, atheromatosis is severe The postoperative occlusion will ruin the graft The diagram emphasizes the importance of adequate, below the knee, exposure of the popliteal bifurcation (By courtesy of Dr Emerick Szilagyi, Henry Ford Hospital, Detroit, Mich)

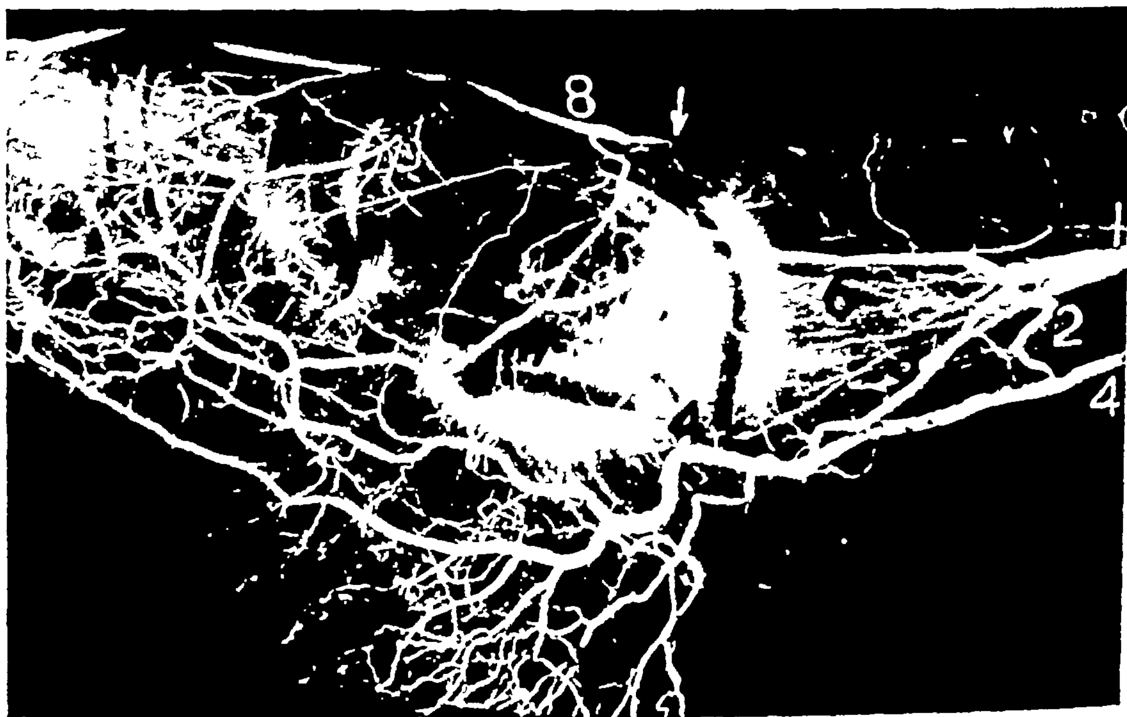


FIG 141 Cadaver angiogram obtained with 75 per cent lead in liquid paraffin The peroneal artery (1) is the main pathway to the foot The anterior (8) and posterior tibial arteries (4) are closed higher up on the leg The peroneal artery connects with both of these and provides an excellent network (Rodda, R Arteriosclerosis in Lower Limbs J Path and Bact, 65 315, 1953)

some other form of evaluation of arterial sufficiency below the knee is urgently needed

Our service has paid considerable attention to this problem, since it is generally agreed that the most perfect bypass or end to end procedure will fail if the distal run-off is poor The aortograms, especially if done serially,



FIG 142. Record of intra arterial pressure of 60/40 mm. Hg obtained in a femoropopliteal segment below a femoral obstruction. This indicates a good collateral circulation and adequate distal arterial tree.

may well visualize the femoral segment it is my preference especially because aortograms seem unnecessary in most of the patients today to do femoral arteriograms at the time of sympathectomy We have also done a number of lower femoral arteriograms (figs 138 139) but these require a splitting of Hunter s canal and when this is indicated it serves a double purpose.

Before deciding on a bypass one can place a 16 gauge needle into the distal segment the rapidity of flow and amount of blood collected in 30 seconds is a fair estimate of the flow distal to the obstruction With the cooperation of Dr Oldrich Prec of our Cardiovascular Laboratory at St Luke s Hospital Chicago we have also taken an occasional intra arterial pressure tracing, and pressures of 40 to 60 mm Hg have been obtained in favorable cases (fig. 142) This is however a lengthy and painstaking procedure and cannot be applied regularly in the operating room There are a number of possibilities to evaluate the distal flow and it would be useful to determine the limit beyond which the defective distal circulation prevents grafting procedures This is the crux of the femoral bypass procedure

Acute Thrombosis in a Previously Stenotic Artery

There are a considerable number of patients showing mild claudication or none at all who suddenly can hardly walk more than a few steps and whose feet become cold, numb and useless As emphasized by Edwards [11] thrombosis is responsible for the major number of acute ischemic catastrophies in the arteriosclerotic limb While atherosclerosis develops slowly and causes a gradual and incomplete occlusion of the artery when thrombosis supervenes—especially at critical levels—the obstruction of the channel is complete and the limb is in danger of being lost

It is for this reason that lumbar sympathectomies should always be performed, so that a better collateral network is available when the occlusion takes place This also holds for patients who have grafts inserted since either the graft or the arterial tree below it may close In addition when an acute thrombosis of an atherosclerotic segment does occur immediate exploration, thrombectomy or endarterectomy should be tried, since a second superimposed thrombus may close important collateral pathways In figure 143 an acute popliteal arterial occlusion is shown in a man with a history of

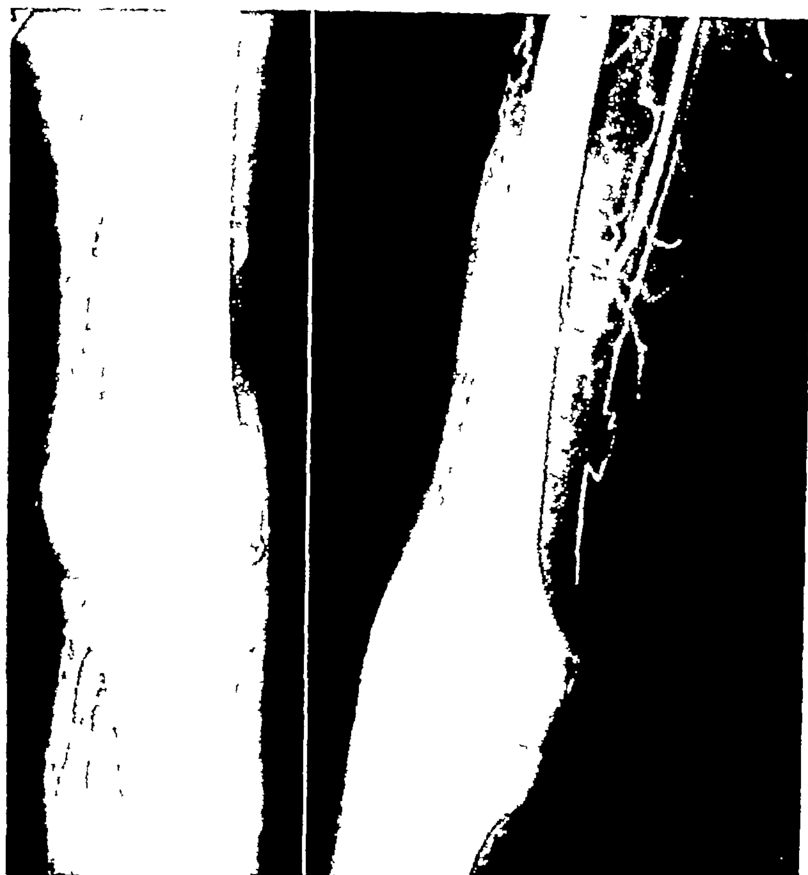


FIG. 143 FIG 144

FIG 143 In the case of E P, a hospital employee, the arteriogram was obtained within 24 hours of the clinical symptoms of an acute arterial occlusion A lumbar sympathectomy was performed followed by four weeks of anticoagulant therapy While the proximal femoral segment looks very healthy, the patient had had previous symptoms pointing to lower leg claudication

FIG 144 Arteriogram of the same patient, E P, who went back to work, but the second thrombus, occurring six weeks after the first, shut off important collaterals so that amputation resulted

many years of claudication Possibly the arteries of the lower leg were involved, but only an emergency lumbar sympathectomy was performed; this transformed his pregangrenous leg to one which he could use for two blocks without having to stop Six weeks later a second vascular occlusion occurred (fig 144), which now necessitated a supracondylar amputation. While the results of thrombectomy in atheromatous occlusions at this level are not always favorable, in this case it might have saved the leg Previous to this experience (October 1952), we had refrained from extracting arterial thromboses if they could be safely differentiated from emboli

Ascending Thrombosis or Granulation Tissue Narrowing or Closing One of the Renal Arteries

This occurs frequently enough to cause concern In figure 145 the atheromatous occlusion of the aorta and iliac arteries is incomplete The patient refused all surgery. Two years later the occlusion is at the origin of



FIG 145



FIG 146

FIG 145 Incomplete aortic and iliac obstruction in a 48 year old telephone operator who refused surgery

FIG 146 High aortic obstruction at the level of the renal artery in the telephone operator (fig. 145) two years later The renal arteries seem still intact The blood pressure rose.

the renal vessels (fig. 146) There was moderate hypertension but no azotemia

The renal arterial stenoses have received much attention recently by Poutasse and Humphries[12] and by DeCamp [13] This problem will be discussed in more detail in chapter 19 Hypertension

Diabetic Lesions of the Peripheral Vascular Tree

A characteristic triad of subcutaneous hemorrhage followed by patches of gangrene increased capillary fragility and anesthesia together with the other neurotrophic lesions, such as clawfoot or Charcot joints are typical of peripheral diabetic lesions Neuropathy will be discussed in chapter 16 Neurovascular Lesions of the Extremities The vascular lesions affect the digital and arteriolar segments and the marked inflammatory reaction seen in many diabetic feet indicates that major arterial supply is usually adequate to cause demarcation of terminal gangrenous areas Arteriosclerosis is of course, present but arteriolar and venular damage is marked The gangrenous heel of the diabetic can be healed by enzymatic debridement and skin graft and multiple toe amputations are better tolerated than in nondiabetic arteriosclerotics (fig. 147) Large areas of cutaneous sloughs can be debrided with streptokinase jelly skin grafts are often successful (fig 148 A and B)



FIG 147 Gangrenous heel in a 50 year old diabetic patient, pedal pulses were absent but the foot was warm It completely healed in three months



A



B

FIG 148 A and B Diabetic arteriolar sclerosis with superimposed infection The large lesion was covered A skin graft was not applied to the submalleolar lesion, which healed much more slowly

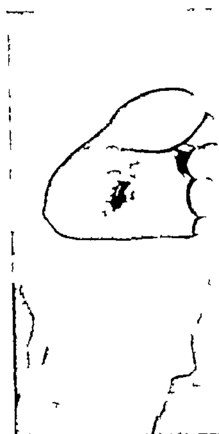


FIG 149



FIG 150

FIG 149 Neurotrophic, painless ulcer leading to the second metatarsophalangeal joint Patient was quickly rehabilitated with amputation of the second toe with the metatarsal head, excision of the perforating ulcer and primary closure of the skin

FIG 150 Incision and drainage of plantar fascia abscess. The fifth toe was removed and the fascia widely split

Equally typical are the neurotrophic ulcers which should be widely excised together with the corresponding toe and metatarsal head (fig 149) Plantar fascia abscesses can be widely drained the necrotic tissue excised and the leg saved (fig 150)

Hypertensive Arteriolar Lesions

This rare but definite clinical entity was independently described by Martorell[14] and Hines and Farber [15] It occurs more often in women usually between the ages of 50 to 70 on the lateral or posterior surface of the lower third of the leg, sometimes surrounded by a purplish halo and showing poor glassy granulations minimal secretion and adherent gangrenous crusts (fig 151)

Our experience agrees with that of Piulachs [16] that neither hypertension nor the presence of pulsations is characteristic of this disease Histologically there is marked subendothelial hyalinosis a thickening of the wall and a diminished lumen to wall ratio but such a reaction is often seen in the hyper plastic arteriolar sclerosis of inflammatory reactions, as in pyelonephritis



FIG 151 Arteriolar necrosis in a 61 year old hypertensive man on the posterior surface of the calf Peripheral pulses were all palpable The large ulcer was covered by a thick hemorrhagic crust Two other patches of hemorrhagic neurosis are seen Healing took eight months

or a postphlebitic induration (fig 152) If the lesions were due to hypertensive arteriolar disease, it is strange that they have not been encountered in advanced, premalignant and malignant types of hypertension (see chapter 19, Hypertension) The clinical impression is that of a *cutaneous infarct*, but the actual occluding thrombi are usually missed

Such arteriolar and venocapillary lesions are, of course, familiar in diabetes and in thromboangiitis obliterans Why they should occur in the lateral or posterior surfaces of the calves is difficult to understand, unless minute trauma is an additional factor Biopsies of skin at a good distance from the gangrenous lesions have not been reported

Monckeberg's Sclerosis

In 1903, this German pathologist described medial calcification in arteries not necessarily harboring subintimal atheromatosis [17] In 130 autopsies he found this to be present in 42 per cent of the cadavers Clinically, however, the pure form of medial calcification is rare, Silbert and his coworkers gave an excellent description of the problem [18] It may occur in the early twenties and there is suggestive connection with a disturbance of calcium metabolism Patients affected with medial calcinosis have night cramps frequently, they may have Paget's disease, renal or submaxillary calculi and

osteoarthritis It is supposed to be frequent in tropical countries and also in the native Negro population of South Africa

There is considerable confusion, both among pathologists and clinicians whether such an entity exists at all In most patients there is additional intimal and subintimal reaction with stenosis or occlusion Occasionally however one encounters a pipe stem artery with its tubular cast of calcification showing transverse ridges instead of coarse longitudinal flakes and no obstruction to flow (fig. 153) An arteriogram in the same patient shows some serration and a short segment of stenosis, but flow is comparatively unimpeded as far as the popliteal bifurcation (fig 154)

Even bone may form in the media and fractures of the artery are possible [18] The surgical significance of extensive medial calcification lies in the fact that such vessels are unsuitable for endarterectomy since there is little if any cleavage existing in the atheromatous core and in the fact that healing of such a vessel to any kind of an implant is slow Rupture of the site of anastomosis from 8 to 10 days after an arterial transplant may be due in part, to the poor vascularization of such a vessel when contrasted to the vascular repair around an atheroma Several times one may back out of a proposed arterial resection when medial calcification is too extensive



FIG 152. Hyperplastic arteriolar sclerosis with venular thickening in diffuse arteriolar disease around a postphlebitic induration



FIG 153 FIG 154

FIG 153 Note the fine, granular transverse ridges and the tubular calcification of both superficial and deep femoral arteries of a 68 year old woman Vessels pulsated well and there was no claudication

FIG 154 Arteriogram of the patient whose flat plate is shown in figure 153 With the exception of a short segmental stenosis, the lumen is patent as far as the popliteal bifurcation

COURSE OF ATHEROSCLEROTIC DISEASE

Although clinical symptoms, especially claudication, give one a fair idea whether or not a patient's atheromatous disease is stationary, slowly progressive or acutely deteriorating, there are many pitfalls in the evaluation of subjective symptoms These can be improved by placebos, by a rise in pressure on coming to a doctor, by vasodilators of the muscular arteries of the epinephrine type, such as Arlidin, and most importantly by actual systematic daily use of the muscles to a point of tolerance We have long noticed that a patient who was forced to walk during the course of his employment, such as a mailman or a bank messenger, improved much faster after a sympathectomy than did a sedentary office worker or a Sunday golfer

Therefore, simple objective tests, such as the venous filling time, the oscillometer or skin temperatures, may give one a far better idea as to how the patient is doing One of course would like to do a number of aortograms and arteriograms to follow the development of obstructions and the collateral pathways, but our service has always showed an active dislike to employing these methods serially.

While the clinical forms just described may be used for grading severity of the disease there are many other factors outside of the ac sites and lengths of arterial occlusion which determine operability and results to be expected from surgical procedures Ever since 1939 Will Beck Eunice Roth and I have used a simple classification into four grade and have pointed out that whether one is evaluating the results of sym thectomy or intermittent venous hyperemia, the stage of the disease in wl these methods are undertaken is of paramount importance in obtai favorable response Many poor results of sympathectomy or arterial graf occur when this simple principle is ignored

Massarelli and Estes[20] have studied 105 patients with clinical manife tions of aortoiliac occlusion especially in regard to the spontaneous co of the disease The ideal candidate for surgery in their opinion is who shows no clinical evidence of coronary or cerebral arteriosclerosis no evidence of diabetes mellitus or other significant systemic disease At all the patient s claudication must be severe enough to warrant the n tality morbidity and expense of aortic resection

The prognosis of intermittent claudication has been naturally of c siderable interest Based on the material one is dealing with (whether patient is clinic or private) mortality loss of limb or serious cardiovasc accidents will occur in a wide variety of instances [21] In Stammers mat nearly all of the patients between 55 to 60 years of age were dead in years in Spaulding s cases 13 per cent were dead in three years Hines Barker reported 59 5 per cent mortality in three years Richards,[21] fi whose article these figures are quoted, found a 28 3 per cent mortality in years from the onset of symptoms Of 17 patients seven died of myocar infarction three of cerebrovascular accident, two of congestive heart fai and a few died of other causes 10 per cent (six patients) needed amputat The point was emphasized that as time goes on the claudicator is les danger of losing his leg than of disability due to coronary or cerebrovasc accident

Throughout this entire monograph I have tried to stress, however importance of individual versus group prognosis In our experience, pati with hypertension diabetes and hyperlipemia are obviously apt to have m progressive vascular damage In the individual case, such prognostic feat are weighed when any type of surgical procedure is advocated

TREATMENT

Physical Therapy

The use of indirect heat, *i e.*, heat applied to the abdomen or to the n has been advocated by us on the basis of venous occlusion plethysmograph While it has been generally recognized that heating an ischemic limb creases pain and accelerates gangrene a whole set of ingenious, therr regulated heat cradles appeared on the market in the 1930 s with the ide

supplying heat, from moderate to 85° F, which eliminates vasoconstrictor tone

Conversely, particularly through the activities of F M Allen, the advantages of a cold environment, even as far as packing the extremity into ice, have aroused great interest [23] While one can advantageously pack an obviously lost extremity into ice and thereby decrease absorption of necrotic and infectious material and relieve pain, refrigeration is definitely condemned in an extremity to be saved There may be some virtue in providing an environment of 50° F in erythromelalgic states, including the hyperemic phase of the immersion limb,[24] one can obtain, however, no advantage in chilling an extremity with impaired circulation For this reason we feel that the Landis-Gibbon test can be used for therapeutic purposes We actually found that if the abdomen, pelvis and thighs were exposed to a heat cradle with controlled temperature, the increased flows obtained in the feet depended entirely on the grades of atherosclerosis, roughly 40 per cent increase in the amount of blood flow per 100 grams of tissue having been obtained [22] This was only slightly less than when direct heat was applied and obviously much less than one finds in an individual with normal circulation Thus, Brown and Allen[25] applied heated sleeves or boots to extremities and obtained an average of 168° per cent rise in an unheated arm with normal circulation

This simple, inexpensive and indirect heating of an ischemic extremity by cradles or electric pads is preferable to indirect heating with diathermy, short wave diathermy or any other form of penetrating heat In considerable experimentation, which included muscle flow studies, with the late Dr John M Coulter, head of Physical and Occupational Therapy at Northwestern University Medical School, the conclusion was reached that the intermittent one hour physical therapy administered to such ischemic limbs by physical therapists was inferior to a continuous 24 hour application of moderate, reflex heat

Attention should be called to the fact, however, that a sympathectomized limb cannot be heated by indirect heat, nor by typhoid vaccine for that matter, since the vasomotor center acts by relaxing vasomotor tone through sympathetic pathways It is also evident now that reflex vasoconstriction or vasodilation will fail to appear when the thermal stimulus is applied to a sympathectomized extremity, since the sympathetics carry afferent, thermoregulatory fibers [26] In other words, the direct, reflex component of the Landis-Gibbon test is abolished while the delayed indirect response due to the warming of the heat center is not interrupted

Thus, for many years indirect heat has been in use on our service Its only limitation is the patient's intolerance to heat It acts by abolishing vasoconstrictor tone

The use of six icebags applied over a sterile towel and surrounded by a rubber sheet and a large turkish towel is used only after the patient has given his consent to amputation. The proposed level of amputation is marked on the extremity and the nursing staff is emphatically instructed to keep the

icebags below the level of the proposed amputation The ice has to be changed about every four hours A moderate dose of narcotic such as 1/6 gr of Pantopon should be given one half hour before the first application, which is painful From then on pain relief is very satisfactory

Intermittent Venous Hyperemia

Following the lead of Collens and Wilensky [27] Hick Coulter and I and later Evoy and I [28] summarized the reasons which prompted us to use this method in the treatment of obliterative vascular sclerosis The rationale of this treatment has remained controversial and very few clinics use the method today Because of the advent of newer and far more potent methods of therapy the place of this simple inexpensive home treatment has become more and more restricted With Evoy it was pointed out in 1948 that diabetic patients with neuropathy patients exhibiting pronounced vascular spasm and those showing arteriolar or capillary stasis are not suitable subjects Patients with diffuse vascular sclerosis unsuitable for sympathectomy or restoration of continuity or those whose claudication remains disabling after sympathectomy constitute the group for whom this simple method may prove useful Here again the earlier phases of the disease showed much better response

It is freely admitted however that with the advent of some of the epinephrine like products which increase blood flow to the muscle and with the recognition that systematic, slow walking whenever feasible is an excellent measure to produce hyperemia in the muscle there has been less occasion to use the mild reactive hyperemia and the mechanical filling and stretching of the venocapillary bed which alternate venous constriction and release produce

A small portable apparatus which delivers constriction from two to eight minutes and release for equal amounts of time is used by the patient in the horizontal position the lower limb or limbs being treated are elevated on one pillow The duration of constriction is determined by watching the subdiastolic pressure (usually 60 mm Hg) produce marked filling of the dorsal veins with rubor of the toes During release, the blood drains out of the veins and the toes must regain their previous color before another cycle of constriction is started

In severe occlusions constriction should not exceed 40 mm Hg Usually an hour in the morning and another hour at night of this treatment is prescribed but one sees patients running this silent apparatus all night, supposedly relieving their rest pain This is an objectionable procedure since it may produce edema. When that is the case the treatment must be stopped or the duration of the cycles and the pressures must be adjusted

While this method of course may act as a placebo some patients have used it for 10 to 15 years and keep returning to it after a period of abstinence Objectively one cannot find any rise of skin temperature or increase in blood flow after a *single* period of treatment nor are there arteriograms

available to show any increase in collateral circulation Since we have never used alternate hot or cold baths nor the postural Buerger-Allen exercises, this seems an adequate substitute and a passive exercise

Other Forms of Physical Therapy

During the course of the years, alternate suction and pressure, oscillating beds, multiple venous compressors producing mechanical massage and many other methods have been studied and clinically evaluated for the Council on Physical Therapy of the American Medical Association It has been evident that any mechanical method designed to increase blood flow must be simple, inexpensive and operated *daily at home* by the patient for many months or years Group therapy of ambulatory patients who come with much effort and considerable expense to get an hour's treatment with an imposing gadget has little justification and resembles group psychotherapy

Drug Therapy

The fundamental objection to the use of vasodilators in regional ischemia has been clearly expressed by DeBakey, Burch, Ray and Ochsner in their paper on "hemometakinesia," the borrowing-lending mechanism.[29] This concept decries the use of generalized vasodilation to improve the circulation of a single ischemic region, since all that happens is that the blood is diverted into normal vascular areas capable of vasodilatation, and away from where it is most needed and where either spasm or organic obstruction will result in less response and less volume flow One can readily observe such a phenomenon when one injects a potent ganglioplegic agent such as tetraethylammonium chloride intravenously to test hypertensive patients (see chapter 19, Hypertension) The extremities become warm in spite of a fall in blood pressure, but should there be an organic occlusion in peripheral or visceral vessels, the limb will become ischemic For this reason, one cannot recommend either oral or injectable vasodilators for the prolonged treatment of peripheral vascular sclerosis This is not to say that in acute arterial occlusions, such as occur during a thrombosis of an atheromatous segment, a potent vasodilator cannot regionally be used In this monograph, I can only mention the drugs which have been in use in our clinics A full description of the medical treatment of peripheral vascular disease has been given by Allen, Barker and Hines [30]

In acute arterial occlusions the use of intravenous and intra-arterial *papaverine* have long been recommended from this institution [31] ½ to 1 gr of papaverine may be injected intravenously in pulmonary embolism or intra-arterially, if a pulsatile vessel is available, proximal to the obstructing thrombus or embolus (fig 73) The local use of papaverine in 2 5 per cent solution, as advocated by John Kinmonth,[32] is most helpful in getting vein grafts to relax or in opening up sutured vessels when they go into a myogenic spasm when intraluminal pressure suddenly rises in them There has been no appreciable benefit, however, from the long term use of oral administration

of papaverine, even in 3 to 4 gr doses daily It simply makes patients constipated and sometimes stuporous

Ronlacol tartrate an alcohol derivative of nicotinic acid dilates the terminal vascular bed mostly the blush areas such as the face and neck It has been widely used in our clinic in 100 mg doses three to four times a day more recently its cholesterol lowering activity has been reported but the doses are huge, 1 Gm or more per 50 pounds of body weight [33] Unless patients complain of itching or an intensive feeling of heat after its use its prolonged intake over a period of months and years is safe although obviously not too much can be expected from it

Ascorbic acid and *hesperidin* in 100 mg doses given three times a day do seem to affect the capillary fragility of hypertensive and diabetic patients not related to low platelet counts The hemorrhagic blisters on diabetic toes and the areas of ecchymosis after minute trauma are favorably affected While the tourniquet test of Rumpel Leede is a very crude method of measuring capillary fragility the improvement in capillary resistance has been definitely measurable after prolonged combined use of these vitamins

Arlidin hydrochloride (nylidrin hydrochloride) an epinephrine like product given in 6 to 12 mg. doses three times a day after meals, is the only vasodilator in use because it seems to have a selective activity on the small vessels of striated muscle and since it does improve claudication unless muscle ischemia is too far advanced It has been known since the work of Allen Barcroft and Edholm [34] that there is a large transient vasodilatation in the *forearm during intravenous infusion of epinephrine lasting no longer than* two minutes after which there is a less intense but sustained rise in blood flow

The rise in blood flow is definitely not due to a rise in blood pressure nor is it due to an increase in pulse rate since atropine can counteract the tachycardia but the increase in flow is still demonstrable While the initial vasodilatation is a direct effect of epinephrine on the terminal vessels in the muscle the sustained vasodilatation which is of clinical interest, has been thought to be caused by the liberation or activation of some vasodilator substance Norepinephrine on the other hand causes constriction in muscle vessels in addition to cutaneous vasoconstriction

These pharmacologic considerations have been discussed in some detail since an effective oral epinephrine like product may well be available for the production of hyperemia in the muscle Thus far this seems to be Arlidin but another drug[35] having possibly less cardiac accelerator effect is now under investigation in our institution

Arlidin is contraindicated in patients with recent coronary thrombosis with cardiac decompensation or with auricular fibrillation It affects an area of the peripheral vascular tree which is inaccessible to surgery

Diets

If atherosclerosis is a metabolic disease which surgeons can only attack at sites of segmental stenosis or occlusion, some thought must be given to a

postoperative regime destined to inhibit further deposition of lipid material in yet uninvolved or slightly involved areas, at the site of autogenous and homologous grafts and particularly at suture lines When plastic substitutes are used, such as Nylon and Orlon, experimental hypercholesterinemia in excess of 1,000 mg per cent will not produce any atheromatous deposits in the graft except at the suture lines,[36] but deposits do occur in frozen homologous arteries Furthermore, as indicated by the experience of Szilagyi and his co-workers, the frozen homologous arterial grafts, especially in the femoral artery, will show much degeneration, atheromatosis and aneurysm formation [37]

Since blood cholesterol determinations are readily available and more easily determined than total lipids, lipoproteins or cholesterol-phospholipid ratios, it has been our custom to follow the patient's blood cholesterol every three months and try to hold it below 250 mg. per cent This can be accomplished with thyroid extract, if basal metabolism or protein-bound iodine are low, and with a low cholesterol diet which contains unsaturated fatty acids and pyridoxine [38] The diet list given all patients who suffer from peripheral atherosclerosis with a cholesterol level above 250 mg per cent is shown in Table IV In addition, linodoxin, containing linoleic (essential unsaturated)

Table IV

LOW CHOLESTEROL DIET

FOODS TO ALLOW	FOODS TO OMIT
Meat or substitutes Lean meat, trim visible fat, fish, poultry, cottage cheese, eggs—3 per week only	*Meat or substitutes* Pork in all forms, corned beef, luncheon meat, smoked or salted meat, canned fish, all whole milk, cheese
Vegetables As desired	
Fruits As desired	
Beverages Coffee, tea, skim milk, buttermilk	*Beverages* Whole milk, any with cream
Bread and Cereals As desired	*Bread* Hot breads
Soup Fat-free broths or those made with allowed foods	*Soup* Cream soup
Desserts Angel food cake, water, ice or sherbet, allowed fruits, gelatin, pudding made with allowed foods	*Desserts* Any containing nuts or cream, ice cream, rich pudding
Concentrated Fats Butter—3 pats daily *only*, corn oil, soybean oil, salad dressing prepared without eggs and with corn or soybean oil	*Concentrated Fats* All shortenings, lard, animal fats
Concentrated Sweets Sugar, honey, jelly, sugar candy, syrup	*Concentrated Sweets* Chocolate candy
Seasonings As desired	*Miscellaneous* Nuts and gravy—unless made with corn or soybean oil as fat, olives, avocado chocolate, mayonnaise, peanut butter cream

fatty acid and pyridoxine given in 2 to 4 capsules three times daily before meals definitely reduces cholesterol levels If in addition the cholesterol lowering effect of Roniacol is widely confirmed,[39] one has done as much as possible to control the lipid imbibition factor which is obviously only one factor in atherosclerosis

Sympathectomy

Our results with sympathectomy in the treatment of obliterative arteriosclerosis have recently been discussed in detail[40] Improvement has been reported to occur in as high as 93 per cent and as low as 50 per cent of the cases Obviously case selection is highly important and the inclusion of patients in whom sympathectomy has been a failure and in whom the operation was done as a last resort before an amputation simply means failure of surgical judgment and inability to size up the limitations of the procedure Recently Smithwick[41] has grouped his patients into twelve groups and used a flushing time of 20 seconds as a criterion for a successful sympathectomy The flushing time is the appearance of dependent rubor and measures according to Smithwick the presence of adequate collateral circulation In our experience the grading of the patients into four groups and the combination of sympathectomy with arterial surgery and possibly with minor amputation makes sympathectomy a simple, harmless and most dependable procedure certainly not to be used only when there is nothing better to do

Actually each individual patient needs a careful study as to whether or not sympathectomy will benefit him. While in the past much use has been made of paravertebral sympathetic blocks rather potent arguments have been raised against the routine use of this procedure At best it can only produce the effects of immediate temporary sympathetic paralysis so that gradual development of collateral circulation is not observed as one sees it clinically over a period of six months to a year Then, also one can never be sure that the block is complete and better results can often be obtained from sympathectomy than what might be expected from a preoperative sympathetic block

For this reason our service has not done routine paravertebral sympathetic blocks on patients for whom clinical judgment would indicate a favorable response The cool blue perspiring extremity without trophic changes in the skin will always react better than the dry hairless extremity with parchment like skin and capillary hemorrhages[42] Much depends of course on what the sympathectomy is expected to accomplish and this needs some discussion in detail

If sympathectomy is done with the hope of increasing walking ability it is quite clear that extremities will respond according to the site and extent of atheromatous occlusion Often in bilateral sympathectomies the one extremity—the less involved one—will respond much better than the other In earlier years we have walked the patients before and after sympathetic block, but this is a misleading procedure since some infiltration of procaine

into the lumbosacral somatic outflow invariably is present and thus the pain of claudication is dampened by sensory interruption

In a most thoughtful evaluation of preoperative laboratory tests, Husni and Simeone[42] found that if sympathectomy for intermittent claudication was only done in patients whose temperature response to sympathetic inhibition was excellent, two thirds of the patients did well Most of these, however, had thromboangiitis, whereas most arteriosclerotic patients failed to improve in his series Our attitude toward the effect of sympathectomy on claudication is as follows one can never promise improved walking ability to any arteriosclerotic patient whose superficial femoral artery or any vessel above this is occluded, because the soleus-gastrocnemius group cannot get much postoperative increase in blood flow, at least right away But it is true that following sympathectomy, reinforced by Arlidin and a low cholesterol, unsaturated fatty acid intake, much improvement can be obtained within the period of a year In the absence of conspicuous muscle atrophy, these patients will continue to walk farther and farther, provided of course an acute arterial thrombosis does not supervene, a hazard for all arteriosclerotic patients

The patchy fibrosis of calf muscles, as seen in gastrocnemius biopsies, may well correlate with the inability to improve claudication both by sympathectomy and by femoral artery bypass

When, however, sympathectomy is performed to prevent or limit the level of amputation, the preoperative skin temperatures obtained on the plantar surface of the big toe after a posterior tibial block (see part II, Methods of Diagnosis) are of decided value While a constant temperature room is of great help, one can do these tests at room temperatures of approximately 70° F (25° C) A "paradoxical drop in temperature," as pointed out in 1944,[43] indicates advanced digital and arteriolar involvement and this occurs not only in diabetes, hypertension and gout, but in atherosclerosis without any obvious additional factor.

Any therapeutic attempt, be it surgical, medical or physical therapy, will run into resistance when arteriolar sclerosis is present Louis G Herrmann made a special point of this in relation to suction and pressure therapy [44] If, therefore, posterior tibial block fails to raise the skin temperature of the anesthetic big toe in a moderate temperature environment, it means one of two things. either organic terminal vascular disease is so extensive that blood cannot reach the toe, or complete sympathetic paralysis is already present One encounters this in certain forms of diabetic neuropathy

Actually, the maximal skin temperature obtained during preoperative testing is practically the same as that produced by sympathectomy.[42] As pointed out in part I of this monograph, these skin temperatures remain high, even although the initially maximal postoperative blood flow settles down to about double of the preoperative level If sympathectomy is done to aid the healing of ulcers, they will heal rapidly unless again the arteriolar type of ulceration is present [14]

It is worth noting that the simple determinations of skin temperature

after posterior tibial block which is available on any surgical service without much technical assistance or laboratory equipment correlates with rates of blood flow obtained by venous occlusion plethysmography or isotope studies Of course if sympathectomy is planned for a pain problem or if the arterial occlusion is very high a high lumbar sympathetic block is to be substituted

In earlier years we had hoped that atheromatous lesions might not progress in sympathectomized limbs but with increasing length of observation one can see that the originally better extremity harboring a short occlusion may go on to widespread destruction as illustrated in figure 155 We still believe strongly however in the principle of prophylactic sympathectomy meaning that should major arterial thrombosis take place in the less affected extremity usually regarded by the patient as being normal the added ischemia will be tolerated with less chance of gangrene [45] A further argument for performing bilateral sympathectomies in obliterating arteriosclerosis is the readily demonstrable presence of cross innervation between the two limbs, so that a more threatened extremity is never as well denervated until the opposite leg is sympathectomized [46]

Since acute thrombotic occlusion may occur in any atherosclerotic limb brought on by mild trauma hypotension or hemorrhage into a plaque this



FIG 155 (A) Arteriogram of E.A. the "good" leg, asymptomatic at this time. The opposite femoral artery had an extensive endarterectomy (B) Four years later the same superficial femoral artery is widely obstructed with no decent distal arterial tree. There is severe claudication. The opposite leg is symptomless, although the endarterectomized segment is closed.

complication certainly holds for patients in whom endarterectomy or a grafting procedure has been performed It has been a definite principle in this clinic to always proceed with a sympathectomy, either as a preliminary stage or at the time of direct surgery for arteriosclerosis, because of the high percentage of late closures after bypass procedures in the femoropopliteal segment In fact, such an extremity whose graft suddenly becomes occluded may be more ischemic than before the bypass procedure This complication will be discussed with vascular grafts, p 236

To sum up, sympathectomy in peripheral atherosclerosis is indicated in the following four grades of the disease [46]

(1) In warm, lower extremities with slight claudication, maintained but diminished pulses, and arteriographic evidence of segmental stenosis or obliteration and diminution of oscillations after exercise. This is a grade 1 arteriosclerosis

(2) In the presence of a cooler foot when the extremities are exposed to room temperature There are no trophic changes There are no pedal pulses, but there is a slight, slow swing of the oscillometer at the ankle indicating a collateral pulse In the arteriogram, the superficial femoral artery is occluded, but the profunda is intact and at least one of the vessels below the knee is patent

(3) In this stage, claudication occurs within half a block, the toes are shiny, atrophic and cyanotic, but the middle portion of the foot and the heel are warm or can be warmed up by vasodilating procedures In the arteriogram such patients show diffuse involvement Saving the patient's limb depends on the profunda and possibly the peroneal artery. Sympathectomy here may have to be combined with high midmetatarsal amputation

(4) This is a pregangrenous or gangrenous stage, in which sympathectomy may lower the level of amputation, much more can be accomplished by sympathectomy on the opposite, less involved side

Finally, all patients selected for thromboendarterectomy or vascular grafts are subjected to a preliminary sympathectomy, prior to or concomitant with these procedures

Side Effects of Sympathectomy

Since this operation, especially lumbar sympathectomy for obliterating vascular sclerosis of the lower limb, has now been so standardized and is so simple, with a negligible mortality and morbidity, I shall limit myself to two complications which do occur and which should be understood

STERILITY OF THE MALE While many authors, including ourselves,[47] have studied this complication, the most thorough and up to date statement of this problem has been given by Whitelaw and Smithwick [48] From experience based on 183 questionnaires turned in by sympathectomized patients, it appeared that preservation of the first lumbar ganglion on one side is a good safeguard against "dry intercourse." Bilateral lumbar sympathectomy from L_1 to L_3 resulted in permanent loss of seminal emission in 54 per cent

and temporary reduction or permanent loss of erection in 63 per cent of the cases

In arteriosclerotic patients especially those with lesions at the bifurcation of the aorta impotence is present to start with in many although by no means in all cases It is in just such a situation that bilateral high lumbar sympathectomies are done and we agree with Leriche Lilly and Grimson that in such high occlusion at least both first lumbar ganglia should be removed [49] In fact, as pointed out under causalgic states (p 442) the tenth to twelfth thoracic ganglia need to be excluded if the entire lower extremity is to be sympathectomized While the value of this low thoracic high lumbar sympathectomy has been questioned [50] there are too many clear-cut instances of improvement when a higher sympathectomy is added to the customary one which does not remove the first lumbar ganglion In patients who have one well pulsating femoral artery the high sympathectomy need only be done on the side of the iliac occlusion While in our hypertensive patients such sterility has occurred it need not necessarily do so [47] The whole question has been recently discussed in more detail [40]

THE POSTSYMPATHECTOMY NEURALGIA Early in our experience in the late 1920 s and early 1930 s about 10 per cent of patients subjected to lumbar sympathectomy complained of genitofemoral neuralgia which lasted four to six weeks and was readily controlled by codeine and aspirin In the last few years this postoperative complaint has occurred in *practically every case* naturally some patients being more vociferous about it than others

The neuralgia does not develop until about the tenth postoperative day and most patients have left the hospital by that time More recently since doing preliminary sympathectomies prior to endarterectomy or grafting, the pain developed right under our own eyes and could be more closely studied The pain or paresthesia is limited to the external surface of the thigh along the distribution of the lateral cutaneous nerve but then again it may radiate down on the medial surface toward the patella as if it had a femoral distribution It does not radiate toward the genitalia and hence could not be a true genitofemoral nerve involvement While the skin may be sensitive the pain has deep boring character an ill localized visceral type of pain

Over the course of the years many possible factors have been given our attention Large Deaver retractors which possibly contused the somatic nerves on the psoas muscle have long been eliminated as the cause Only small Hibbs sponges are used to retract the psoas the vena cava or the aorta While Cushing s clips have been applied to the severed trunk and some of the rami, the neuralgia still appears when they are eliminated Procaine has been injected to the proximal and distal segment of the ganglionated trunk during sympathectomy While the vasoconstriction in the foot occurring at the time of section or clipping of the trunk can be thus abolished,[51] the neuralgia is uninfluenced

The possibility of activating a lumbosacral arthritis or a trochanteric bursitis by the patient s position on the table has been considered But the neuralgia also appears after transabdominal sympathectomies with a small

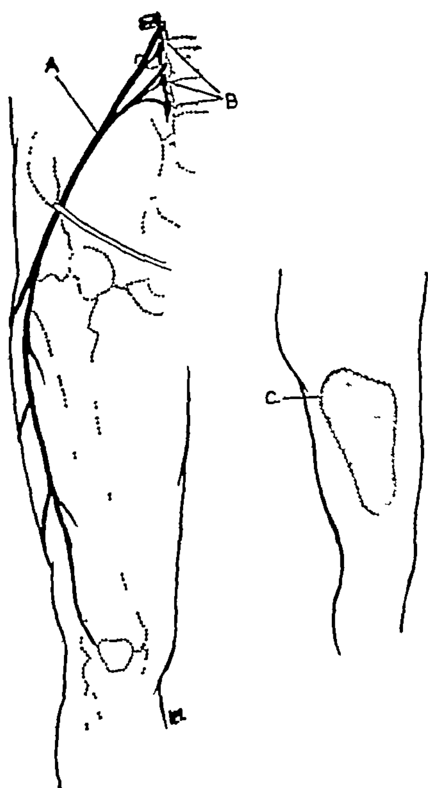


FIG 156 Course of the lateral cutaneous nerve of the thigh (A), its relationship to the ganglionated trunk (B), to Poupart's ligament and to the fascia lata Partial injury to the nerve may produce both pain and hyperhidrosis in the corresponding cutaneous segment (C) denotes the cool, moist, paresthetic area

pillow under the patient's back Besides, this neuralgia has no resemblance to the radicular pain of osteoarthritis Nevertheless, in a few cases Passler's suggestion of manipulating the spine was followed,[52] with no relief In one patient the orthopedic department injected the trochanteric bursa, but this failed to give relief

Since spinal anesthesia leads to a certain small percentage of neuralgias, a large series of sympathectomies have been performed under general anesthesia No decrease of neuralgic pain was evident

There finally remain two mechanisms which are now under study and which, of course, should they be valid, have therapeutic implications One observation was made by Tracy and Cockett,[53] that the painful paresthetic cutaneous area is usually moist and cool and shows the kind of marginal hyperhidrosis which one usually sees at the border of dry sympathectomized skin My associates and I, in grappling with the problem of intercostal neuralgia after thoracic sympathectomies, have tried paravertebral blocks central to the denervated area years ago, and, while there is occasional success, we have not used it consistently to be sure enough At the lumbar level, injecting the lowest dorsal segments paravertebrally has not been tried often enough, but it has failed in some instances

The second possibility is that of a traumatic neuritis of the lateral cutaneous or femoral (and not genitofemoral) nerve, which is especially apt to occur when the section of the chain is at the crus of the diaphragm This nerve originates from the undivided primary rami of L^2 and L^3, but from their posterior divisions, and is notoriously vulnerable both at the inguinal ligament and where it perforates the fascia lata on the thigh (fig 156). The syndrome of meralgia paresthetica is well known and much effort has been

made in the past by neurologists and orthopedic surgeons to treat it The nerve contains sympathetic fibers and hence the area is cool and moist just as in any irritative lesion of a mixed nerve One can often see a cool moist skin in a sciatic syndrome

Considerable space has been given to this complication and its possible management, since some prominent vascular surgeons notably Michael De Bakey[54] are turning to low sympathectomies to avoid the neuralgia and thus are not able to obtain the benefit derived from high lumbar sympathectomies

If the patients are told about this complication ahead of time if they do not fear the onset of some vascular or neurologic accident if they are told that it is annoying, innocuous and will subside in four to six weeks, most of them will bear it during the day perhaps needing narcotics for the first few weeks The use of 1 mg. (1 000 micrograms) of vitamin B_{12} widely employed by thoracic surgeons for the post thoracotomy neuralgias has been more or less disappointing

If paravertebral block is going to be used it should be used early a few days after the onset of pain just as in a causalgic pain the longer it is allowed to persist, the more it resists treatment

Thromboendarterectomy

For those of us who regarded an injured intima as one of the most potent factors of thrombosis, the technique of endarterectomy seemed like a mysterious procedure I saw one of the early French films on the subject and felt that it was a highly traumatizing incomplete and objectionable method, and had not the slightest desire to try it Actually by 1950 Arnulf[55] had collected 40 cases with 8 deaths and 17 patencies, which was a little over 50 per cent success in the surviving patients The method got a slow start in this country but the California group pioneered by Norman Freeman[56] and the experience of Richard Warren in Boston[57] showed immediate results which deserve attention Our clinic did 12 endarterectomies in 1954 seven in the aortoiliac and five in the femoropopliteal segments These were re-examined at this writing in the spring of 1958 This was purposely done on a small material and closely followed so that the limitations of the operation could be well defined.

The original description by Cid Dos Santos[58] called for short, incisions of 2 cm. in the artery With the help of a blunt spatula, not unlike the one used in submucous resections in the nose the inner core of the closed vessel was reamed out leaving a bluish, translucent striated wall which consisted of adventitia and part of the media It is to the credit of Dos Santos that he recognized such a line of cleavage of which no pathologist has ever heard The French surgeons notably Reboul,[59] spoke of a sequestrum a dead inner core from which the vascular outer wall was readily separated by blunt dissection This group incised the entire length of the obstructed artery instead of making multiple short incisions, each one of which might be the future cause of stenosis A forced freeing of the lumen has been tried before notably

in 1884 by the Rumanian surgeon Severeanu who catheterized a pl arteriosclerotic artery with a fine urethral sound during an amputatic obtained a free pulsating flow of blood at the cross section of the stu

All of the early cases of endarterectomy were followed by ge heparinization of the patient, with the idea of keeping the reamed se free until some sort of nonwettable lining developed The study of these reveals much overdosage, hemorrhages and sloughs, a condition whic has plagued some of the earlier grafting procedures

In spite of our initial reaction against this procedure, it seemed to many advantages, such as (1) utilizing the patient's own arterial tree patent channel, (2) not destroying any of the collateral supply, which is surprisingly patent when the obstructing core is removed with its initi 3 mm. plug in the collateral, and (3) being a much more simple and sl procedure than excision and end to end grafting This last advantage, ever, is not as definitely applied to the bypass procedures, which can be almost as rapidly

The first obvious disadvantage of the method is that once the p line of cleavage is established it is hard to know when to stop, sin thickened, atheromatous intima and subintima can be followed centrall peripherally for a great length Second, with extensive calcification wall, there may remain nothing at all but a translucent adventitia, as as 1950, Wiley and his associates advocated wrapping the weakenec with fascia lata [61] This problem of a porous versus nonporous mater an arterial substitute will receive further consideration when these a prostheses are discussed (p 549) Aneurysms after endarterectomy actually been reported Third, since some mobilized thickened intima remain distal to the extracted core, the greatest care must be exerted to s it back to the remaining outer layer, since dissection of blood distal t arteriotomy may readily close the distal segment and produce retro clotting of the reamed artery

Our own small experience limited to seven aortoiliac and five f ropopliteal endarterectomies has led to the adoption of the following ir cations for this type of operation (1) In the absence of severe calcificati massive lymphatic reaction around the artery, a short unilateral or bil common iliac stenosis or occlusion can be successfully reamed out, pro the "saddle plaque" at the aortic bifurcation is clearly enucleated I external iliac artery is badly diseased, this operation is apt to fail, anc happened to two of our iliac endarterectomies (2) In the femoropop segment, where the atheromatous stenosis is much more extensive tha occluded segment in Hunter's canal would lead you to believe, unles entire segment from the femoropopliteal to the common femoral artc reamed out with the Cannon stripper[62] immediate or early closure o segment is bound to take place As will be illustrated in part IV, Sur Technique, a transverse division of the occluded segment at its lowest sible level will yield the best line of cleavage for the stripper and perm accurate distal fixation of the loose intima to the arterial wall All of

limited endarterectomies done in this location in 1954 had closed although the patients did not require or request any additional surgery They were all sympathectomized (3) While the patients received from 30 to 50 mg. of heparin into the distal stump and into the systemic venous circulation no patient was heparinized postoperatively because of dire results observed in the hands of several surgeons, starting with Leriche

While opinion and experience has still not stabilized it would seem that endarterectomy is useful in short common iliac stenoses or occlusions the long iliac occlusion including the common femoral *probably* does better with a bypass As in all surgical techniques enthusiasm and meticulous care will yield better results in the hands of those who perfect a technique and stick to it For this reason further long endarterectomies using the technique of Cannon and his associates[62] are still under trial on our service (fig 157)

To sum this up at present, endarterectomies are done (1) for short common iliac stenoses or occlusions with enucleation of the saddle atheroma at the aortic bifurcation and (2) for short well localized femoral occlusions but not if widespread medial calcification is evident in the flat plate The distal segment may have to include the popliteal bifurcation, however, where so much disease is found



FIG 157 This inner core of the superficial femoral artery stretched out, was 25 cm in length, and ranged in diameter between 10 and 15 mm. It consisted basically of masses of fibrin, hyalinized fibrillar tissue and acidophilic granular precipitates with many circular clefts, as if a lipid material had been dissolved.

Excision of the Occluded Artery and Graft

This procedure has already been discussed in the treatment of arterial injuries in which it is eminently successful since the closure is localized and the artery above and below the traumatized vessel is intact The atherosclerotic vascular tree constitutes a much greater test of this prodecure and, as pointed out in the description of the clinical forms of the disease the *single segmental occlusion* is obviously the ideal case To indicate the rapidly shifting tendencies in vascular surgery this procedure in spite of its obvious short comings, was widely heralded and practiced until a few years ago when the bypass procedures largely supplanted it below the inguinal ligament

Excision of an occluded artery (arterectomy) had been advocated in the past by Leriche and Stricker in their monograph [63] The observation that vasodilatation occurred following the removal of a closed arterial segment, provided sufficient collateral circulation developed, led to the conclusion that a localized sympathetic denervation and elimination of vasomotor reflexes was at play I shall not describe the many attempts reported in the literature on total excisions of the superficial femoral artery and on excisions of the aortic bifurcation, which later gave way to short segmental resections said to relieve the longitudinal contraction of the artery

On the vascular service at Hines Veterans Hospital, Philip Shambaugh tried a series of femoral arterial resections for the rest pain of the ischemic arteriosclerotic limb, but there was no obvious improvement. Looking back on these interesting studies of Leriche, it seems today that arterectomy, just as periarterial sympathectomy, may have sectioned some sympathetic fibers which the customary lumbar sympathectomy did not interrupt With the use of the high lumbar or dorsolumbar sympathectomies these periarterial fibers are sectioned, and the operation of arterectomy as such has fallen into a well deserved disuse

Another principle which nature undoubtedly uses to enhance collateral circulation should be mentioned here. This is ligation of an occluded artery just below its main collateral pathway, so that the pulse wave can be thrust undivided into the collateral channel Actually, Dean D Lewis in 1927 advocated the ligature of the superficial femoral artery to enlarge the profunda in cases of thromboangiitis obliterans [64] This mechanism is seen in action when one exposes the femoral bifurcation and sees a huge profunda femoris originate from the common femoral artery The profunda shifts from its posterior position and lies flush and parallel with the occluded superficial femoral artery Section of the occluded superficial femoral artery at this level *might* prevent an ascending thrombosis, but this procedure has not been carried out during femoral arteriograms when the opportunity might arise

The obvious step after excision of an occluded arterial segment was to replace it with a vascular or synthetic substitute, the experiences with aneurysmal resections have already been described (p 140) In occlusive disease, such as the full-blown Leriche syndrome, various degrees of occlusion of the lower segment of the abdominal aorta and both iliac arteries can be successfully excised and grafted with both homologous frozen bifurcational segments or with plastic prostheses The relative merits of these substitute materials will be discussed in part IV, Surgical Technique According to Michael E DeBakey's overwhelming experience, 95 per cent of the occlusions of the aortic and iliac arteries and over 50 per cent of the occlusions of the femoral arteries can be reconstructed [65] Charles Rob, on the other hand, finds that out of 822 patients studied for atherosclerotic occlusion, about 50 per cent of the patients with occlusion of the abdominal aorta and about 25 per cent of the patients exhibiting femoral arterial thrombosis are suitable for direct reconstructive procedures [66] As pointed out by DeBakey in the Year Book of General Surgery for 1957-1958,[67] variations in the material may well bear on

his discrepancy My associates and I have been extremely careful in establishing the indications for this procedure possibly due to conservatism, to a respect for the natural course of the disease and no doubt due to lack of large experience which no amount of armchair strategy can supplant It has been of great interest to compare the management of the last 50 patients seen in my private practice with the last 50 patients seen in the Vascular Clinic (Table V)

Table V

MANAGEMENT OF 100 CONSECUTIVE PATIENTS WITH ATHEROSCLEROTIC VASCULAR DISEASE

NUMBER OF PATIENTS	NO SURGERY	SYMPATHECTOMY*	ENDARTERECTOMY	GRAFT	AMPUTATION
A 50	15	30	5	12	5
B 50	20	17	1	2	10
Total 100	35	47	6	14	15

A Private patients
B Clinic patients
* With grafts and endarterectomies or alone

There is a marked difference in the handling of these two groups Some might expect the more radical treatment in a teaching institution where new methods are continuously being tested On the contrary because of the earlier appearance of patients in private practice than in the clinic more radical procedures can be done for them with more chance for success The opportunity to see this divergent material is a small addition to the argument for part time professors, should it need any Thus 60 per cent of the private patients had sympathectomy against 40 per cent of the clinic patients 10 per cent of the private patients had endarterectomy against 2 per cent of the clinic patients 24 per cent of the private patients had grafts against 4 per cent of the clinic patients and 10 per cent of the private patients required amputation against 20 per cent of the clinic patients

All this really amounts to the fact that atherosclerosis is a progressive disease and if one could now state that by earlier reconstruction the disease has been halted, a more vigorous attack would seem in order especially on the so-called good leg, the asymptomatic leg But we do not even know that the low cholesterol diet with unsaturated fatty acids is going to stop further atheromatous deposits For this reason direct surgery for arteriosclerosis [68] a term first coined by Ormand C Julian of our institution must be regarded as a palliative measure to overcome a threatening localized obstruction independently of what might and does occur in the future

The limiting factors of excision and grafting are (1) severe involvement at and above renal vessels in the abdominal aorta, although a limited endarterectomy can free this segment and permit a clamp below the renal ar

teries, (2) insufficient runoff at the distal segment, as evidenced by poor or absent filling of a well timed arteriogram or by poor back pressure of blood in the distal segment, which one can occasionally measure with a manometer, (3) severe multiple occlusions distal to the proposed resection or elsewhere in the body, and (4) poor cardiac, cerebral, renal or hepatic reserve, indicating short life expectancy.

The excision of the occluded segment in the aortoiliac area is a long, often difficult procedure, periaortic lymph glands are much in the way and some of the aortic wall may have to be left behind, matted to the vena cava While most of the collateral lumbar vessels are closed, some are unnecessarily ligated and sacrificed in this procedure In the femoral segment, a lengthy dissection and excision of the closed superficial femoral artery certainly sacrifices collaterals, traumatizes the saphenous nerve and may lead to sartorious claudication or slough as a result of injuring vessels to the muscle The bypass procedure to be discussed next overcomes these objections to a considerable degree

For these reasons, the excision of the closed segment has gradually given way to the bypass procedures first conceived by Kunlin and Leriche in 1949,[69] with a slow but later thunderous acceptance in this country.

Bypass Procedures

Robert Linton[70] has given a great impetus to the use of end to side vascular anastomoses Both he and DeBakey's group[71] have pointed to the advantage of creating large anastomoses by this method, which actually creates a new large collateral vessel and leaves the occluded segment with some of its collaterals undisturbed In addition to the same limitations which prevail when excising and bridging the occluded segment, thought must be given to the danger of creating a bypass in the presence of incomplete occlusion, unless one is willing to ligate below the bypass Gangrene of the extremity, or at least aggravation of ischemia, may result when the current of blood is diverted into *two channels*, both of which may suddenly close It has also been rightly pointed out that existing collaterals which have slowly developed in the presence of a gradual occlusion, may cease to function in the presence of a fresh new channel, should this channel close, the acute occlusion will find the circulation unprepared to carry blood to the periphery [72]

Nevertheless, a conservative employment of the bypass procedures both in the aortoiliac and in the femoropopliteal segments seems to be the simplest and most promising approach to the reconstruction of atherosclerotic vessels Here again the state of the segment distal to the occlusion is of paramount importance, and is to be examined not only by arteriograms but by direct exposure of the critical segments, namely, the femoral bifurcation and the popliteal bifurcation Whereas the first obstruction can still be bypassed by the iliac to the lower femoral segment, the state of the popliteal and the tibioperoneal segment is so important that one must explore them prior to any side-tracking or reaming procedure

In part IV under Vascular Prostheses (p 548) the problem of the type of material to be used for grafting will be discussed

General Summary of the Surgical Treatment of Atheromatous Occlusions

On our service every patient showing evidence of obliterative vascular disease of the extremities whether this is segmental or diffuse is offered a *sympathectomy* This is done not so much to improve claudication—although it will do so in early segmental occlusions—but as a protection against trophic changes or gangrene

When there is a short segmental stenosis or occlusion in the lower abdominal aorta and particularly in the iliac arteries *endarterectomy* is the method of choice Whether endarterectomy is preferable here to a short bypass is debatable and has yet to be determined Endarterectomy in the femoropopliteal area has been a failure in my hands but the question has been reopened again with more extensive reaming, better instruments and newer techniques

Excision and *grafting* in occlusive disease have been simplified and made less hazardous to collaterals by the *bypass procedures* which are the methods of choice in (1) the full blown Leriche syndrome with high aortic involvement (2) the femoropopliteal area when the presence of a sufficient outflow tract is definitely established

That no surgical procedures are indicated for patients with marked cerebral cardiac renal or generalized vascular damage or for those with a short life expectancy due to malignant disease should need no emphasis This rule however is frequently broken by some whose technical skill exceeds their knowledge of the natural course of the disease or their ability to size up the patient s bodily and emotional response to stress

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CHAPTER 11

TUMORS OF THE VASCULAR SYSTEM

THE VASCULAR SYSTEM INCLUDING VESSELS OF ALL SIZES AND THE LYMPHATICS is a common source of benign tumors but a relatively rare source of malig nant ones There is a multiplicity of nomenclature and the American Cancer Society records 224 terms for the tumors of this group In chapter 7 Congenital Vascular Anomalies attention was drawn to the fact that improper development of the complicated vascular network often leads to cutaneous nevi to vascular masses, and to multiple arteriovenous fistulae but these are not tumors in the histologic sense of the word and have already been dis cussed. To distinguish between malformations hamartomas and benign neoplasms of blood vessels is often impossible

CLASSIFICATION

A growth may develop from any element of the vessel wall and hence one can encounter a hemangioendothelioma a vascular leiomyoma and a hemangiopericytoma Their recognition calls for an expert pathologist For the vascular surgeon the important lesions to recognize differentiate and treat are the glomus tumor Kaposi s hemorrhagic sarcoma and the lymph angiosarcomas in the lymphedematous extremities I shall confine myself to the description of these three entities since the recognition and management of the various hemangiomas and lymphangiomas would take us into the realm of tumor surgery

These lesions may occur in any part of the body Excellent descriptions of them may be found in an atlas published by the Armed Forces Institute of Pathology[1] and in the writings of Arthur Purdy Stout [2]

The Glomus Tumor

In chapter 2, Vascular Shunts, the wide distribution of vascular shunts between arterioles and venules and their rich nerve supply as described by Masson,[3] were discussed The painful subcutaneous tubercle of Wood (1812) was probably of this nature [4] but credit should go to Masson for the histologic description of the painful subungual growth which has now been seen by many authors including ourselves

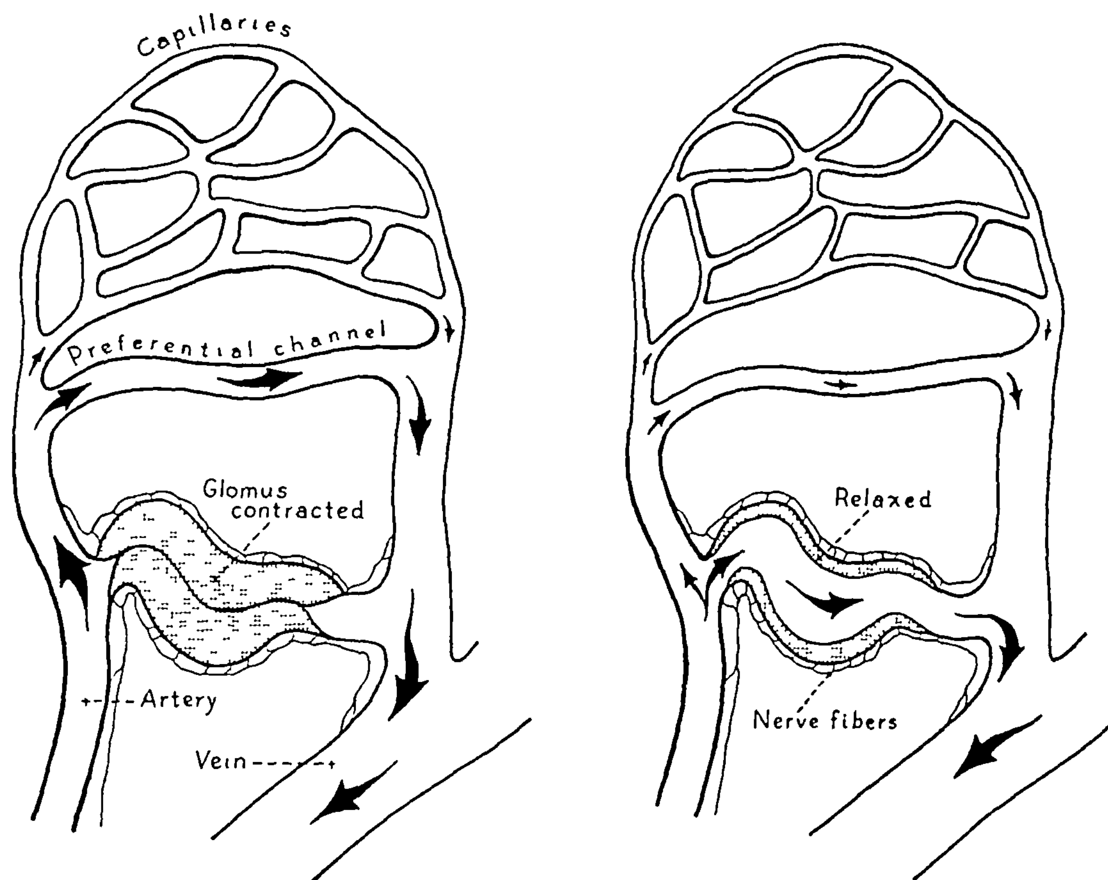


FIG 158 Diagram of a normal glomus in a contracted and relaxed state The coiled, corkscrew-like connecting channel, richly supplied by myelinated and nonmyelinated nerve fibers, is the origin of the glomus tumor See figure 4 for the hemodynamic significance of these channels

The neurovascular glomus is found overwhelmingly in the distal part of extremities, particularly under the nailbed, but it has also been described in the visceral organs The tumor itself originates from the coiled, corkscrew-like channel which connects a small artery with the subpapillary venous plexus This Sucquet-Hoyer canal is surrounded by loose connective tissue and is richly supplied by myelinated and nonmyelinated fibers (fig 158) It is characteristic of this arteriovenous anastomosis that it has no elastic fibers and no circular muscle fibers, but contains large pale staining polyhedral cells, "epitheloid" cells Masson originally described three types of tumor, the angiomatous, the cellular and the neuromatous, depending on the tissue prevailing in the histologic sections The lumen may be surrounded by numerous normal glomera and many Pacchionian bodies, which are known to register pressure It is known that glomus tumors are exceptionally sensitive to both external and intravascular pressure changes (fig 159)

Clearcut trauma is elicited in about half of the cases, there is a definite *tendency to tumor formation in many patients* [5]

The tumor appears as a tiny bluish spot, under the nail it has a greyish appearance It is exquisitely tender and when gently touched with an applicator may bring on a paroxysm of pain radiating up to the neck and shoulder

In the angiomatous type widespread vascular changes not unlike those of the first stage of causalgia are encountered The entire hand or forearm is warmer the veins are prominent and the oxygen content of the venous blood is increased Reflex dilatation of the peripheral vessels may be responsible for this [6] since it is difficult to conceive that the tiny arteriovenous fistula would have such a diffuse hemodynamic effect This is the explanation given by Katz and Goetz,[7] who demonstrated dilated and tortuous radial and ulnar arteries with an arteriogram in the case of a glomus tumor under the nail

In a personal case the glomus tumor was not detected and the patient was subjected to a dorsal sympathectomy because of the painful throbbing causalgic state of the hand which followed a minor crushing injury The vasodilation and the diffuse pain disappeared but the localized pain led to the detection of a pinpoint sized bluish spot which was readily excised withs complete relief of pain

Associated with the vascular changes, widespread sweating, increase in growth and osteoporosis have been described One must differentiate a glomus tumor from causalgia, from hemangiomas which are pink and pain less, from melanomas angiosarcomas and neuromas and from many skin tumors None of these show the exquisite pain on pressure and cause the diffuse vascular changes of the glomus tumor Goetz,[8] in his excellent de scription of the glomus tumor cites the case of Blumenthal whose patient



FIG 159 (A) A glomus tumor with two vascular channels lined by normal endothelium but surrounded by "epitheloid" cells with clear cytoplasm. (B) A more cellular part of the tumor with palisading cavernous channels. (By courtesy of Edwin F Hirsch, Department of Pathology St Luke s Hospital Chicago)

was treated for 13 years for angina pectoris and whose angina was cured by excision of a subungual glomus tumor of the left ring finger

Complete excision of the growth will cure the condition Recurrences have been reported in the literature, but these are due to incomplete removal A malignant degeneration is unknown

Kaposi's Hemorrhagic Sarcoma

One might wonder about the inclusion of this lesion here, but in my experience it is not infrequently encountered This low grade malignancy



FIG 160 Small, raised, hemorrhagic papules in an elderly man suffering from Kaposi's sarcoma The lesions slowly progress toward the groin and metastasize (From the Department of Dermatology, U of Illinois College of Medicine Reproduced from a Kodachrome slide)

occurs mostly in males (94 per cent of 434 collected cases)[9] and usually in the fifth to seventh decades The disease is most often seen as red macules or papules of no characteristic shape on the lower extremities It spreads slowly and proximally, creates hard, poorly movable inguinal lymph glands and leads to visceral metastases in the gastrointestinal tract, liver, lung and spleen Gastrointestinal bleeding is perhaps the most common cause of death Occasionally the primary lesion is in the viscera, with late skin involvement or no skin involvement at all

The reddish lesions later become bluish-brown or black and their micro-

scopic appearance is most confusing since early lesions look like a capillary or cavernous hemangioma Older lesions progress to greater degrees of cellular proliferation and fibrosis, at which time the vascular channels are obliterated In the end stages this process looks like a fibrosarcoma or a neurofibrosarcoma Spontaneous hemorrhages are common in the earlier stages and confusion with a purpura is possible Different stages of this slowly invasive and metastasizing process may be encountered in the same patient In one paper[9] 28 synonyms are listed for this well vascularized fibrosarcoma or sclerosing angiosarcoma Death occurs in from 1 to 10 years

In a personally observed case of a 72 year old man an initial diagnosis of thrombophlebitis was later changed to lymphedema caused by inguinal metastases The local dark lesions at the ankle were reported as capillary hemangiectasia and a biopsy of the lesion sent to a skin pathologist was reported as a benign vascular fibroma. Only having seen this man once in consultation the slow progress of the lesion was observed through correspondence Eight years after his visit to Chicago he was still active in Florida real estate The lesion is so characteristic that, having seen it once it is hard to miss in spite of a negative or inconclusive pathologic report (fig 160)

There is no effective treatment known but roentgen ray treatment is customary

Lymphangiosarcoma

This is a rare lesion and is difficult to distinguish from other sarcomas including Kaposi s sarcoma The majority of the lesions have arisen in arms affected by a postmastectomy lymphedema after a long, latent period of 5 to 25 years This is a nodular friable hemorrhagic mass the tumor cells are spindle shaped invade the affected tissue diffusely and palisade around blood vessels to form small clefts[1,10,11] Certainly these tumors are difficult to distinguish from other sarcomas and the suspicion has been entertained that they are undifferentiated carcinomas[1] We have seen retrograde carcinomatous metastases in the lower extremities after a carcinoma of the cervix with inguinal metastases producing a lymphedema while this is a clinical entity worth recognizing, it may not have a uniform histologic picture

Other Tumors

There are many other forms of benign endothelioma or hemangioma which occasionally become malignant and may then be called angiosarcomas It is important but often difficult, to differentiate these from congenital arteriovenous communications which spread, increase in size and erode bone but do not show any cellular proliferation. Excellent histologic pictures of these can be seen in Landing and Farber s monograph[1]

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CHAPTER 12

THROMBOEMBOLISM

THROMBOEMBOLIC PHENOMENA OCCUR WHENEVER THE VASCULAR TREE IS occluded by thrombi in situ or by emboli from a forward or backward thrust of thrombi to distant parts of the arterial or venous circulation In this chapter attention will be focused on venous and arterial thrombi and emboli of the extremities Pulmonary embolism, as a consequence of peripheral venous thrombosis will also be discussed While it will not be possible to cover thromboembolic phenomena in visceral organs brief mention will be made of these for two reasons First they are basically identical with those seen in the periphery and second they often accompany or complicate peripheral vascular thromboembolic lesions and therefore need recognition A recent small monograph on thromboembolism gives a more detailed sur vey of the subject [1]

PREDISPOSING FACTORS

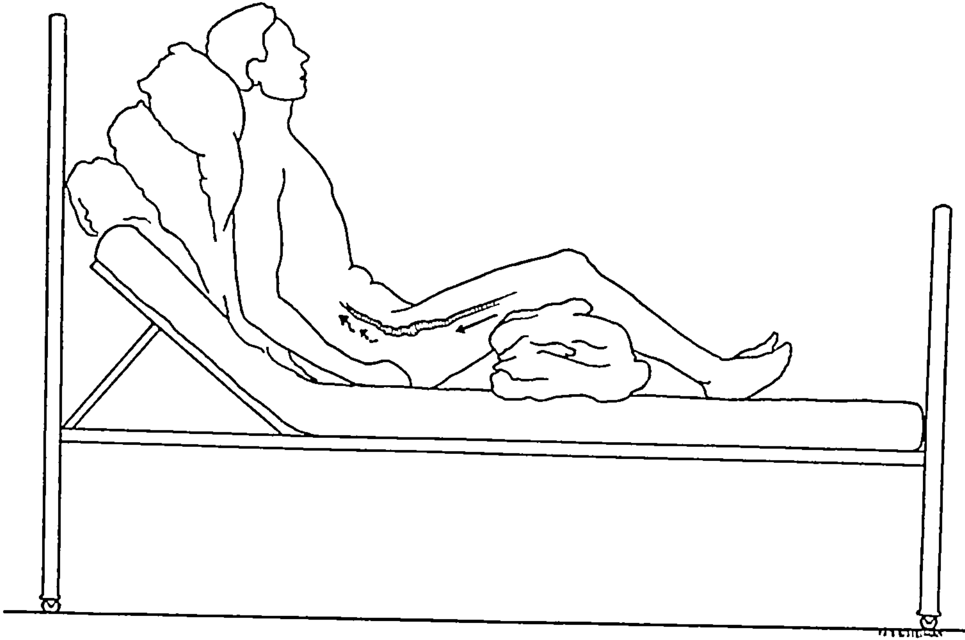
Slowing of circulation, changes in the vessel wall and disturbances in the clotting mechanism are the three factors first emphasized by Virchow in the pathophysiology of intravital clots In part I Fundamental Principles Affect ing Vascular Surgery these factors have already been discussed in detail (pp 26-35) It is sufficient to say here that an interaction of these factors is necessary for intravital clot formation, and that while on the venous side a decrease in velocity of flow is the dominant factor for the localization of a thrombus on the arterial side a stenosis coupled with sudden hypotension is frequently recognizable Since methods of prevention are based on the recog nition of such factors they will be now discussed

PREVENTION

Prevention of Venous Thrombosis

Development of stasis in certain areas of the venous bed may be combated by simple measures directed toward increasing the rapidity of venous return These consist of the following. (1) *Elevation of the foot of the bed 6 to 8 inches* which is only contraindicated in congestive heart failure or in spreading intraperitoneal infection. This measure can be used postopera tively after childbirth or in prolonged bedrest It is far superior to the cus-

FIG 161



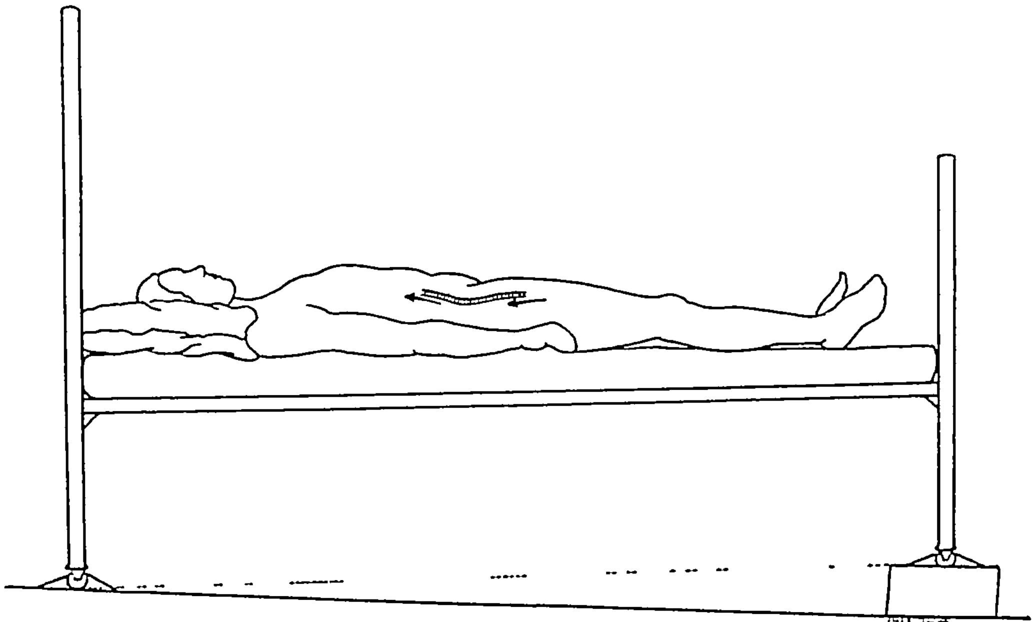


FIG 162

FIG 161 The customary Fowler's position in bed kinks the main venous channel at the knee and groin (de Takats and Jesser Pulmonary Embolism J A M A, *114* 1415, 1940)

FIG 162 Elevating the foot of the bed on 6 to 8 inch high shock blocks improves venous return of the lower extremities and pelvis (de Takats and Jesser Pulmonary Embolism J A M A *114* 1415, 1940)

tomary use of pillows under the foot and lower leg, which produces kinking at the knee and groin and does not drain the pelvic veins at all (figs 161, 162) (2) *Frequent flexion and extension of toes and ankles;* these aid venous return and improve muscle tone (3) *Early ambulation* whenever possible, but this does not mean sitting immobilized in a chair with the feet hanging down, since, from the standpoint of venous return from the lower extremities, this position is worse than lying in bed (4) *Frequent periods of deep breathing* for the aspirating effect on the venous pools of the pelvis

In order to accomplish all this, encouragement reassurance and ı but continuous pain relief is important since otherwise the patient will be willing to move the muscle pump

Prevention of Pulmonary Embolism

SUPERFICIAL FEMORAL VEIN LIGATION This has been carried out ‹ large scale, particularly by the vascular surgical service at the Massachu: General Hospital [2] Division of the vein at this level will eliminate all emt phenomena from the plantar veins and calf muscle plexuses It will not vent propagation of thrombi from the adductor muscles through the ‹ femoral vein and it may itself create a common femoral thrombosis b: ascending ligature thrombus It does not prevent pelvic thrombi from br ing loose *we have never used this procedure*

COMMON FEMORAL VEIN LIGATION OR DIVISION This is our rou procedure during all major amputations of the lower extremities [3] Divi of the vein at this site effectively inhibits all embolic phenomena coming f the veins of the lower extremities It creates a permanent edema of the lc third of the leg and the foot but amputation stumps of the lower leg above the knee do not show any but a transient edema While pulmoı embolism is not too frequent after amputations an omission of this ı guard may lead to fatality The father of an internationally known phy: ogist was subjected to amputation and suffered a fatal pulmonary embo on the fifth postoperative day Femoral vein ligation was omitted to sho the procedure in this frail, handicapped patient Under spinal anesth one can often amputate above or below a knee while another team ligates common femoral vein Anticoagulants are not used following amputat since stump hematoma leads to painful fibrosis and interferes with the w ing of a prosthesis

LIGATION OF THE VENA CAVA This operation leads to a permaı interference with venous return and creates a postphlebitic syndrome venous pressure in the leg does not fall on exercise as it does in the noı extremity (fig. 163) The operation should not be employed unless ver thrombosis is inadequately controlled by anticoagulants and has prodı pulmonary embolism, or if bilateral iliofemoral thrombosis can no treated with anticoagulants and an embolus or emboli have occurred. (traindications to anticoagulants will be discussed below

PREVENTIVE USE OF ANTICOAGULANTS A tremendous literature grown up around this subject and it would not be possible to discuss it in detail Basically however the problem revolves around defining an *dangered group of patients* who judged from past experience show ar creased evidence of thromboembolic phenomena Such consideration course can only be applied to postoperative cases postpartum cases such patients who have had a thrombotic episode in the brain heart or o visceral organ

It would be far beyond the scope of this monograph and my experi to discuss prophylactic anticoagulant therapy following coronary thromb

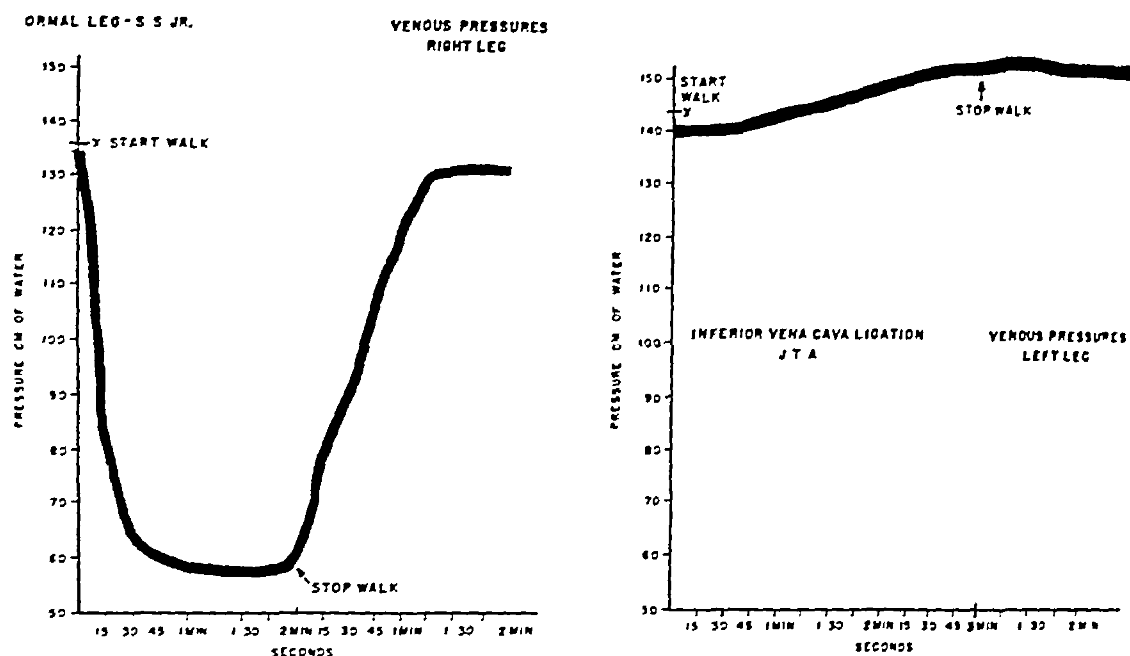


FIG 163 Venous pressure measurements at the ankle when walking, following vena cava ligation Note the absence of fall in venous pressure on the right following a ligation of the vena cava, normal response is on the left (Courtesy of Dr J H Schneewind, Dept of Surgery, U. of Illinois College of Medicine)

or cerebral thrombosis. The only point to be made in this connection is that a differentiation between thrombosis and acute coronary or cerebral insufficiency must be made, and this is not always possible Thus, in the postmortem studies of Hicks and Warren,[4] occlusion of sclerotic vessels by thrombosis was absent in 60 per cent of cerebral infarcts. Equally impressive are the postmortem studies reported by Evans on patients with a diagnosis of acute myocardial infarction, since in well over 50 per cent of the cases brought to autopsies with a diagnosis of coronary thrombosis it was absent This means that 1,358 patients would have received long term unnecessary therapy, had all patients with a diagnosis of coronary occlusion been put on anticoagulants [5]

One does have, however, a much more definite idea about surgical or obstetric patients The insult here which initiates thrombosis is single, its timing is known and one can precede and follow this single insult with prophylactic doses of anticoagulants

In part I, Fundamental Principles Affecting Vascular Surgery, the process of blood clotting was discussed in detail (pp 26 35) The principle was stressed that intravascular thrombosis—not in its microscopic, but in its clinical form—is a decompensation of the clotting mechanism Prophylaxis of thrombosis, then, must consist of supplying the normal anticoagulants and possibly fibrinolysins to the body so that it can cope with a single stress.

For this reason, in a group of endangered patients in whom the incidence of thrombosis following surgery or childbirth is statistically high, prophylactic therapy can be used Cardiac, carcinomatous, diabetic, hyperlipemic, hypotensive, polycythemic and previously thrombotic patients be-

long to this group Voigt and I[6b] showed that such patients clotting mechanism shows a diminished or absent response to the stress of ACTH and therefore needs prophylactic anticoagulants We have used 200 mg. of heparin in two divided doses a day for three days before operating, assuming that we can effect storage of heparin Histologic evidence of such storage in the mast cells is now available [6a]

Another method of anticoagulant prophylaxis is to administer heparin or a coumarin derivative after surgery or childbirth Such prophylaxis usually starts 48 hours after the initiating stress In our study of the postoperative stress on the clotting mechanism the third or fourth day seemed to be the critical period at this time the normal anticoagulant factors come into play [7]

The object of prophylactic administration of anticoagulants is to keep the clotting time slightly above normal levels In R.S a 57 year old woman who has had multiple embolic episodes from a fibrillating rheumatic heart a hysterectomy had to be performed (fig 164) She made an uneventful recovery without hemorrhagic or thrombotic episodes the clotting time was kept between 10 and 20 minutes by intramuscular injections of heparin after an intravenous priming dose

Finally one can also operate on heparinized patients using intermittent or continuous intravascular injections, provided protamine sulfate in a 1 1 ratio is available In thromboendarterectomies and arterial grafts one can

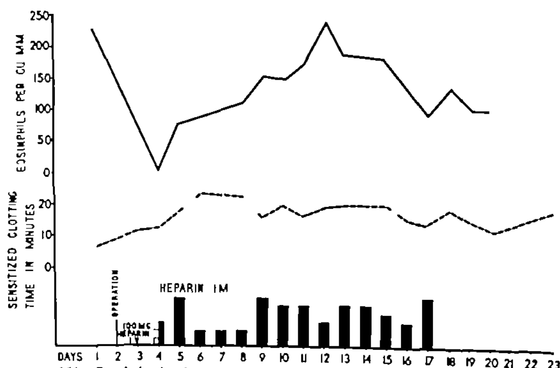


FIG 164 Prophylactic administration of heparin in the case of R.S., a 57 year old woman with rheumatic heart disease, auricular fibrillation and multiple previous emboli to the extremities and to the brain She required a hysterectomy for bleeding due to fibroids. After a priming dose of 50 mg. of intravenous heparin, the clotting time was raised to between 16 and 20 minutes and was maintained by varying the dose of intramuscular heparin between 200 and 100 mg. daily The clotting time was sensitized with 1 gamma of heparin to 1 cc. of blood.

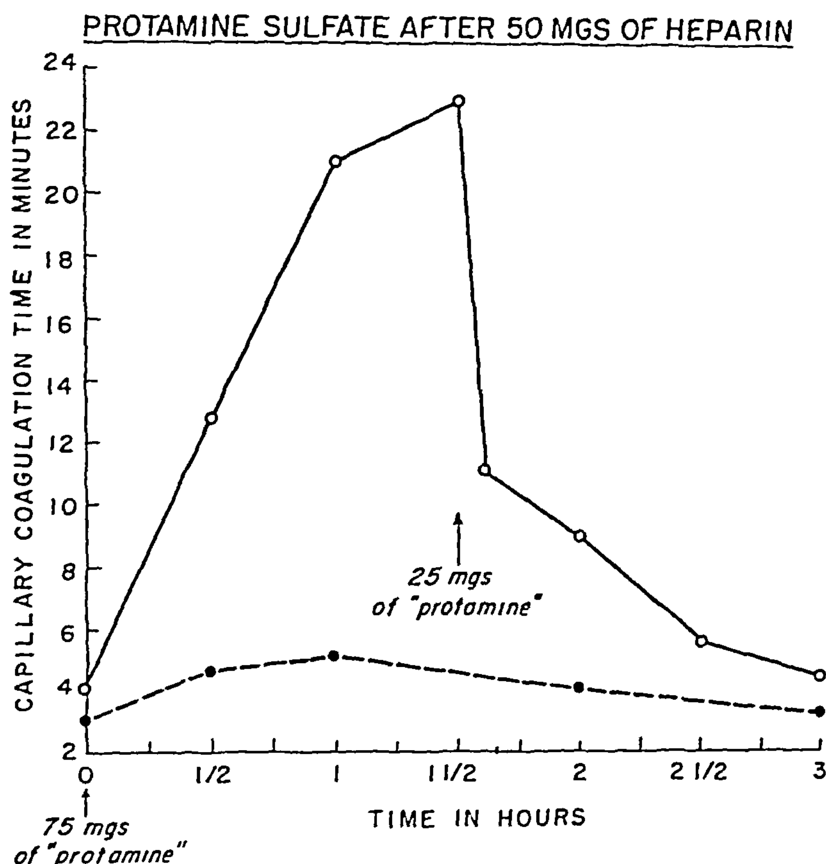


FIG 165 25 mg of intravenous protamine sulfate readily drops the clotting time at the peak of response to 50 mg of intravenous heparin Interrupted line, 75 mg of protamine given simultaneously with 50 mg of heparin, completely neutralizes the effect of heparin The ratio of 1 5 mg of protamine to 1 mg of heparin has been found to be completely suppressive [9]

use 50 to 100 mg of heparin given intravenously Regional heparinization by a continuous intra-arterial drip into the *distal segment* of an artery to be resected or bypassed has been advocated by Freeman and his associates.[8] We have used this excellent method many times, adding 50 mg of heparin to 250 cc of physiological saline solution. One often has difficulty, however, with clotting above the *proximal clamp*, for this reason the administration of 50 to 100 mg of heparin into the intravenous drip has been most useful, with an occasional control of the Lee-White clotting time which is held at 20 to 30 minutes

Regarding this *operative prophylaxis*, very little difficulty is encountered with hemostasis, far less than with postoperative prophylaxis which we do not use after vascular sutures Since this principle has also been recently utilized in cardiac surgery, especially during open dry field procedures, 10 per cent protamine sulfate must be ever present to readily restore the clotting time (fig 165)

For the control of heparin activity, capillary coagulation time, Lee-White single tube clotting time and the sensitized clotting time have been widely used Although we have largely abandoned the use of capillary coagulation time, it is still a useful though crude bedside test, and, as any simple experiment shows, it follows rather closely the more sensitive heparin-

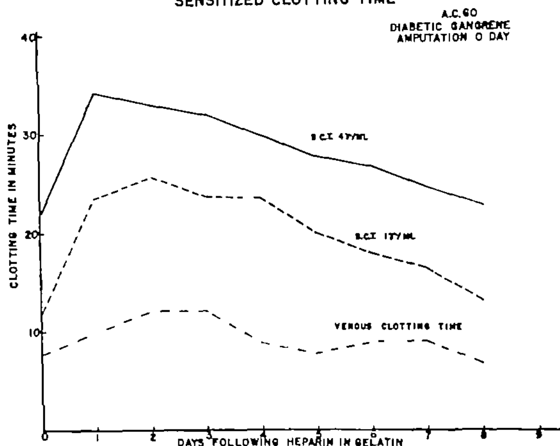


FIG 166. Sensitized clotting times with 4 and 1 gammas of added heparin to each cubic centimeter of blood after a single dose of 200 mg. of heparin in gelatin with vasoconstrictors Note the demonstrable effect of heparin by the sensitized clotting time extending as long as eight days. (de Takats, G Sensitized Clotting Time. Angiology *1* 317 1950)

retarded clotting time [9] The latter is far more sensitive than the customary Lee White single tube clotting time as shown in figure 166

While any laboratory can set up the simple procedure of the heparin retarded clotting time for practical purposes of controlling heparin response the capillary coagulation time is sufficient even although this statement is regarded as heretical by hematologists and anticoagulationists

CLINICAL FORMS OF THROMBOEMBOLISM

Silent clots, clinically unrecognizable may form in any part of the vascu lar tree They may occur in the brain, in the heart in the lungs and in the peripheral arteries and veins They are short clots with sufficient collateral circulation so that symptoms and signs of ischemia or congestion do not occur

Their significance lies in the fact that they serve as a nidus for the propagation of the clot. This happens (1) when blood pressure falls, (2) when direct trauma to the vessel cocurs or (3) when an increased tendency to clotting develops as after operation or infectious disease

Generally speaking, the venous clot will produce an increase in venous

pressure, with resulting edema and often capillary hemorrhage The arterial clot will produce an anemic or hemorrhagic infarct, depending on available collaterals A combination of arterial and venous thrombosis is usually poorly tolerated, and an ischemic leg due to obliterating arterial disease may become much less viable when a venous thrombosis supervenes

1. Venous Thromboses

Cerebral Venous Thrombosis

A typical course of events consists of hemiplegia following puerperium accompanied by convulsions, papilledema and a minimal amount of blood in the spinal fluid. In most of the cases of obstetrical longitudinal sinus thrombosis there was evidence of venous thrombosis elsewhere in the body J P Martin[10] was the first to designate the vertebral venous system as the ascending pathway to the cerebral venous sinuses Two personally observed cases had transitory cord systems

Obviously, anything that will diminish cerebral edema is worthy of trial, such as concentrated albumen or a cervical sympathetic block In one of our cases treated by sympathetic block, a complete functional recovery was obtained from a massive hemiplegia and aphasia Anticoagulant therapy is unadvisable when venous outflow is obstructed in the central nervous system, because of the presence of petechiae and coalescent hemorrhages

There is, of course, a type of ascending thrombophlebitis from infections in the ear, nose, throat and sinuses[11] which has nothing in common with the above mentioned picture, and for which antibiotics and anticoagulants are in use One of our first experiences with heparin was on a well known surgeon who developed orbital edema following a carbuncle of the nose Fever and spreading thrombosis promptly subsided after the administration of heparin

Vertebral Vein Thrombosis

Coelho and I[12] recently presented three case histories which contained the following sequence of events· (1) venous thrombosis of the lower extremities or pelvis, (2) a history of marked strain on or compression of the lower abdominal organs or vena cava, and (3) the appearance of cord symptoms of unknown origin The last was characterized by transient spinal block with a gradually receding level of paraplegia In one case, a laminectomy revealed a nonpulsating dura mater

Since the report of Batson,[13] the role of the prevertebral venous plexus in the spread of carcinomatous metastases and pyogenic infections has been repeatedly emphasized, however, spinal cord lesions following iliofemoral or pelvic vein thrombosis have not been reported The visualization of the prevertebral venous plexus is possible by injection of an opaque substance into a lumbar spinous process [14]

The diagram of venous drainage from the spinal cord is shown in figure

167 A lateral view of this circulation can be seen in the roentgenogram shown in figure 168 There is a noticeably poor filling of the prevertebral plexus between two well visualized lumbar veins but it is notoriously diffi cult to interpret phlebograms In this patient the level of the cord lesion was between L_2 and L_3

It is strange that with the hundreds nay thousands of iliofemoral and vena cava thromboses cord lesions have not been registered There are some other sudden possibly vascular, phenomena in the spinal cord which could be related to the vertebral venous system The cord damage following the use of Efocaine a long lasting local anesthetic used for intercostal nerve block is usually explained by a perineural spread in the spinal cord It could be due however to the propagation of a thrombosis of an intercostal vein to the prevertebral plexus The paraplegia following splanchnicectomy for hypertension reported by Voris and his associates[15] has been interpreted as being due to an anterior spinal or anterior radicular artery thrombosis How ever intercostal veins are often injured and clipped during this operation while we have not encountered this complication after more than 600 splanchnicectomies undertaken for hypertension such a case should be in vestigated by intraspinous visualization and laminectomy

The treatment of this lesion is unsatisfactory Anticoagulants may pre

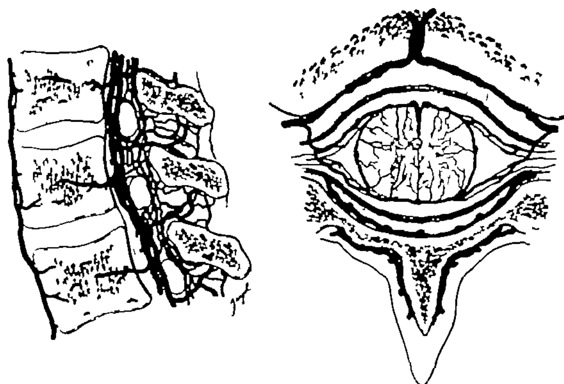


FIG 167 The venous system of the spinal cord. Drainage from the spongiosa of the vertebral bodies and spinous processes occurs toward an anterior and posterior longitudinal vein. There is a well developed vertebral venous plexus, whose connections have been thoroughly investigated by Batson.[13] On the right, the anterior and posterior intervertebral and spinal veins are diagrammatically shown. (Redrawn from Netter Ciba, Inc.)



FIG 168 75 per cent Diodrast has been injected into the spongiosa of the lumbar spinous process The needle is seen in the left lower quadrant An extensive venous pattern is visualized, including two lumbar veins There seems to be a collateral circulation around a poorly filling segment corresponding to the third lumbar vertebra The patient's cord symptoms were at this level

cipitate hematomyelia Paravertebral sympathetic block was done in one of our cases with no effect Neurosurgical exploration was done in another but could demonstrate no spinal block

Venous congestion, produced by ligation of a posterior radicular and a posterior spinal vein in the monkey, produces a complete flaccid paraplegia, which then recedes to a paraparesis which is more severe on the side opposite the ligation At autopsy the spinal cord remains normal to the naked eye, but in microscopic sections scattered fiber degeneration is seen in the anterior, lateral and posterior white columns [16a] The lesion thus is irreversible and permanent Transitory signs and symptoms of spinal injuries may be interpreted as being due to partial venous occlusions

Visceral Venous Thrombosis

Thromboses of the mesenteric, splenic, hepatic and portal veins exhibit characteristic symptoms and are beyond the scope of this monograph However, in widespread thromboembolic disease they do accompany and complicate vascular phenomena in the periphery, and thus a few simple clinical observations leading to their recognition seem to be in order



FIG 169 W M a 55 year old man, had a history of chronic alcoholism and repeated hemorrhages from esophageal varices. A bilateral subcutaneous pattern of veins is visible which is both "caput Medusae" and collateral circulation due to vena cava obstruction. No shunt procedure was possible.

When a patient with known peripheral thrombotic phenomena develops abdominal pain distention hematemesis or bloody stools mesenteric venous thrombosis must be thought of * If the patient s white count and platelet count drop with evidence of some hemolysis and the spleen becomes pal pable splenic vein thrombosis is to be suspected When the patient who has a known vena cava thrombosis develops ascites gastric hemorrhage and a caput Medusae type of venous pattern on the abdominal wall a propaga tion of the thrombus obstructs the hepatic vein with development of Chiari s syndrome (fig. 169)

Venous Thrombosis of the Upper Extremities

Superficial venous segments are frequently occluded as a result of intra venous therapy especially when hypertonic or irritating solutions are used

* While the diagnosis of mesenteric thrombosis is difficult enough, a differentiation between an arterial and venous thrombosis is even more so except that the venous thrombosis starts more insidiously and gradually increases in severity Direct surgical attack on acute occlusions of the superior mesenteric artery has been recently reported.[164]



FIG 170 The number 10 player, Charles B , developed an acute axillary vein thrombosis immediately following this game in 1941 Surprisingly enough, axillary vein thrombosis does not seem to occur often after basketball games, although the stretch and abduction of the arm is formidable

Sometimes a surprising ascending periphlebitis develops, indicating that infection has been carried in or a pre-existing latent infection has been activated. The bland clots often recanalize making venipunctures again possible, and the periphlebitic exudates are readily absorbed In patients suffering from a vascular sensitization, a simple venipuncture may result in thrombosis; this is especially true in thromboangiitis obliterans, where a slight trauma often results in segmental phlebitis

The injection of large, poorly sharpened needles into the dorsal veins of the hand preceding anesthesia often leads to large painful hematomata and thrombosis, in the elbow, a median nerve causalgia due to leaking dextrose solution is not unknown

Axillary Venous Thrombosis

This condition is usually of sudden onset following violent unaccustomed exercise, affecting the right upper extremity more often than the left There is, however, a form which appears gradually overnight and first manifests itself on arising [17] In our personal experience, we have seen axillary thrombosis occur after cranking an old Ford, after grinding spark plugs, after a strenuous swimming match, after a basketball game (fig 170), and after washing windows with elevation and abduction of the arm Rotation of

the arm with full abduction such as turning a screwdriver with the arm above the head or painting a ceiling, may also bring on this vascular accident [18]

Whether a daily occupational abduction of the arm at work would have etiologic significance is debatable but it naturally has some medicolegal implications Such patients should they exhibit axillary thrombosis should certainly have a change of occupation to avoid recurrence

As a result of a sudden effort, the arm swells and becomes slightly cyanotic and pain is experienced There may be a sharp pain under the axilla where a cord is often palpable or there may be a diffuse visceral type of pain tingling and numbness accompanied by or caused by reflex vasoconstriction Collateral circulation which develops over the anterior chest wall and the upper arm becomes visible in a few days The cubital vein is distended and this distention may be increased by exercising the arm Exercise will raise the venous pressure and produce a type of intermittent claudication [19]

An infra red photograph shows more numerous and more distended superficial veins on the affected side (fig 171) A phlebogram may show spasm or complete obstruction of the deep venous channel (fig. 172) Neither of these two procedures however is necessary for the diagnosis

The mechanism of this sudden venous occlusion is unclear it is likely that several different mechanisms may be at play Compression of the vein between the clavicle and the first rib pressure on the vein by the pectoralis minor by the costocoracoid ligament by the subclavius muscle and by the



FIG 171 Infra-red photograph of Mrs. E.L., who exhibited a subacute left subclavian vein thrombosis following a streptococcus sore throat and some muscular effort The venous plexus is more prominent on the left, especially around the nipple.



FIG 172 Phlebogram of a young man who suddenly developed pain and swelling of the right arm during basketball practice After an injection of 20 cc of 35 per cent Diodrast into a prominent cubital vein, complete block of the third portion of the subclavian vein just below the clavicle became apparent, together with marked distention of the axillary segment (de Takats in Christopher's Textbook of Surgery)

subscapularis, and strangulation of the vein by an abnormally placed phrenic nerve have all been suggested (fig 173) Hughes[20a] reviewed these factors and concluded that no single theory could account for all cases. It is certain that a sudden movement with associated muscular contraction traumatizes the vein Thrombosis frequently propagates proximally and distally and much of the permanent disability will depend on the extension of thrombosis As a matter of fact, subclavian vein thrombosis would be a more reasonable diagnosis, and all our roentgenologically visualized cases showed obstruction at the subclavian level

The diagnosis of axillary thrombosis can hardly be missed when the sudden edema after effort, the purplish-blue hand with maintained pulsations and the characteristic collateral circulation are taken into account When undiagnosed and untreated or indifferently treated, there remains a permanent edema with a feeling of tingling, heaviness and cold sensitivity Use of the arm for heavy or steady work will bring on pain with visible increase in venous pressure

Over a period of years, several methods of treatment have been employed In the first case, seen eight months after the initial pain and edema,

the axillary vein was exposed below the clavicle and since the fibrosed c extended into the subclavian a perivenous stripping was performed 1933 [0b] This abolished the vasomotor phenomena and the pain but sc residual swelling remained (fig 174)

Gradually, the following three phasic treatment has been developed the patient seen early within a week or two following the axillary ven block A cervical sympathetic block is performed which invariably creases edema and improves circulation Next the patient s arm is suspen on a Balkan frame and kept there for four or five days until all edema disappeared This is uncomfortable and the patient may require sedati and narcotics Finally anticoagulant therapy is instituted with hepa given preferably through a continuous venous drip into the dorsal vein of affected arm 200 to 300 mg of heparin are given daily into 500 cc flasks (per cent dextrose solution When the arm is removed from the frame the travenous drip is discontinued and a snug elastic bandage is substituted fr the base of the fingers to the axilla Heparin is continued by deep sub taneous injections giving 1 cc. of 10 per cent solution twice a day for other 10 to 14 days

Such an aggressive multipronged attack is justified by the observat that if axillary venous thrombosis is not treated early and intensively pern nent edema and considerable disability with pain on exercise cold sensiti

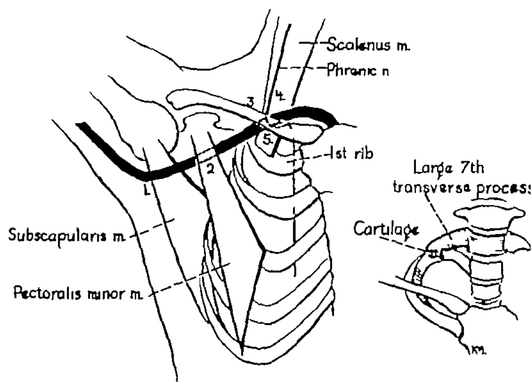


FIG 173 Mechanisms of axillary vein compression, responsible for axillary vein thr bosis The axillary subclavian venous segment may be compressed by (1) the subscapul (2) the pectoralis minor (3) the clavicle, (4) the prevenous phrenic nerve and (5) the scale anticus. The insert shows the cartilaginous extension of the large seventh transverse proc (Modified from Hughes, E. S R. Venous Obstruction in Upper Extremity Brit. J Su 36 155 1948)



FIG 174 Infrared photograph of H S , a well built, muscular man, who, while grinding the spark plugs of his car, was suddenly seized with pain in the right axilla This was followed by swelling of the arm, a deep boring pain after exercise and a widespread arterial flush The subclavian-axillary venous segment was stripped Note the increased venous collaterals on the right (de Takats, G Reflex Dystrophy of Extremities Arch Surg , *34* 939, 1937)

and extensive collateral varicosities remain I have also exposed and aspirated some axillary clots which often extend into the subclavian vein, but such a procedure prevents intensive heparinization, which seems important

Late cases seen months or years later may show vasomotor phenomena seen in minor causalgic states, and after preliminary diagnostic sympathetic blocks the patients are subjected to dorsal sympathectomy Sections of the anterior scalenus muscle and the pectoralis minor tendon have been suggested by Lord and Stone [21]

Superior Vena Cava Syndrome

Obstruction of the superior vena cava may be caused by trauma, chronic adhesive mediastinitis, thrombophlebitis, constrictive pericarditis, aortic aneurysm and pressure of neoplastic growth, such as Hodgkin's disease, lymphosarcoma or carcinomatous invasion, in short, anything in the mediastinum that will compress, kink, encircle and ultimately obliterate the venous drainage from the head, neck and upper extremities

An army sergeant was suddenly exposed to considerable air pressure during battery practice when 12 shots were simultaneously fired He was knocked down, but did not lose consciousness Ever since that time his head and neck became purple-red on bending forward and a collateral network of veins had developed on the anterior chest wall, with dilated cutaneous venules and edema of the eyelids A service connected disability was acknowledged [20b]

The signs and symptoms of obstruction of the superior vena cava are obviously dependent on increased venous pressure in the head and upper extremities. Headache, vertigo, throbbing and blurring of vision, together with venous engorgement of the head, neck, anterior chest wall and upper extremities, are characteristic. In contrast to a subclavian or axillary vein thrombosis, unilateral venous hypertension (innominate obstruction) or bilateral venous hypertension (superior vena obstruction) is present in the neck. It is important to determine whether the obstruction is above or below the juncture with the azygos vein. When there is a prominent venous plexus over the sternum connecting with branches of the internal mammary vein, the venous return is through the intercostal veins into the azygos, and the obstruction is likely to be above the juncture with the azygos vein. Should the obstruction be below the azygos system, the blood must return to the heart through the inferior vena cava, and the collateral veins are visible at the costal margin. When a venous segment is compressed by spreading two fingers and milking out the blood between them, release of the higher finger will produce rapid filling, indicating the direction of flow.

Obstruction of the superior vena cava or of the innominate vein (more



FIG. 175 Phlebogram of a superior vena cava obstruction. The subclavian and innominate vein are seen, together with the superior vena cava, in the case of a mediastinal mass suspected of being an aneurysm. The compression was due to Hodgkin's disease. No intrinsic lesion of the superior cava is seen.

frequently the left because it is longer), may occur in certain cases of h failure, but here the venous pressure is equally elevated in the lower extr ities Terminally, one sees superior vena cava or innominate vein thromb in decompensated cardiac patients Determination of venous pressure phlebography are important diagnostic measures (fig 175)

Depending on the cause of the venous obstruction, the course of disease may be benign or may rapidly lead to death For eight years in vascular clinic we have watched a young Negro girl who developed an asy tomatic pattern of characteristic venous circulation following an attack respiratory infection The important consideration is whether the c pressing lesion is benign and removable, or whether it is due to an aneur or to malignancy. Mediastinal decompression and stripping of the vein h been successfully accomplished [22] In treating thoracic aneurysms by wi and sternal splitting in the preresection era, I have successfully relie superior vena compression in two instances [23] The direct approach, lowing Gerbode's pioneer animal experiments,[24] is an atriocaval shunt a vascular graft between the innominate vein and the pericardial portion the superior vena [25] Such operations have now been successfully perform and while the suitable cases are few and far between, the patients may entirely freed of the disabling venous hypertension of the head, neck upper extremities A number of patients have to be explored before a suita case, nonmalignant, progressively disabling and technically feasible, can found

Venous Thrombosis of the Anterior Thoracic Chest Wall

Described first by Mondor,[26] this manifests itself in palpable, pain subcutaneous cords in the chest wall, frequently thoracico-epigast branches, and gives rise to much confusion and anxiety I have observe number of such patients, women in their forties, who showed no evidence deep venous obstruction, no pulmonary lesions and no involvement of t breast Differential diagnosis from traumatic fat necrosis, lymphangitis a carcinoma of the breast with lateral spread must be made Typically, it co sists of a long cord external to the breast which is rather painless and qu superficial, it is seldom surrounded by periphlebitis, except at the start, a there are no glands in the axilla or groin Occasionally an entire cutaneo network may be involved, giving rise to a reticular pattern of enlarged ha cords

The lesion runs a benign course and may leave some pigmentation, ju as a superficial phlebitis on the lower extremities does When the lesi follows some operation on the breast, such as removal of a benign cyst, malignant lymphatic spread may be readily suspected. The biopsy of such cord will reveal a thrombosed vein with little if any periphlebitic reaction various stages of organization When there is periphlebitis, retraction of t skin may be observed.

Since "Mondor's disease" may occasionally be part of a migratir

phlebitis anticoagulant therapy after careful search for a possible cause of increased coagulability may be considered

Thrombosis of the Inferior Vena Cava

Inferior vena cava thrombosis most commonly results from ascending thrombosis in the iliofemoral segment However external pressure of neoplastic lymph nodes inflammatory processes in the retroperitoneal space abscesses under the diaphragm or an occasional large retroperitoneal hema toma may produce obstruction with secondary thrombosis The result is of course an edema in both lower extremities and a characteristic collateral pattern of subcutaneous veins in the abdomen (fig. 176) In addition vena cava ligation itself may produce an impairment of venous return, although the caval or iliac thrombosis for which it is undertaken has already produced a bilateral postphlebitic syndrome of the lower extremities and the caval ligation may not increase the pre-existing venous hypertension

The subcutaneous collateral varices however are only one of the path



FIG 176 Infra red photograph of an old vena cava thrombosis with well developed col lateral circulation. In Wm. B this developed after a severe attack of typhoid fever " Often these "infectious diseases are really ascending venous thromboses with pulmonary emboli.



FIG 177 35 per cent Diodrast has been injected into a superficial varix on the thigh. In addition to large tortuous varicosities in the abdominal wall, a large collateral channel enters the partially canalized common iliac vein The trunk of the vena cava is closed, but an elongated lumbar ascending vein joins the lower end of the azygos (Case of Ira M , post-traumatic vena cava thrombosis)

ways through which the blood from the lower extremities and the pelvis returns to the vena cava above the obstruction

In the case of Ira M , who suffered extensive shrapnel wounds and multiple fractures at the Anzio beachhead, extensive varicosities are seen on the side of the injection in the abdominal wall (fig 177) An important deep collateral pathway leads, however, through the lumbar veins to the azygos There are also channels from the pelvic veins to the portal system and the vertebral venous plexus may serve as a pathway of decompression

It is well to keep these pathways in mind since they may explain the following clinical observations

(1) Closure of the varices of the abdominal wall by recurrent attacks of phlebitis does not increase venous stasis in the periphery A number of patients exhibit these attacks of collateral phlebitis, but they seem to be harmless and result in disappearance of such varices They do not produce emboli

(2) Hemorrhoids and vaginal varices often follow occlusion of the vena

cava Ascites and portal hypertension frequently are manifested by vari cosities at the outlet of the pelvis

(3) A small group of patients already referred to under the heading of vertebral vein thrombosis has fleeting neurologic phenomena mostly of a sensory nature, which may be due to varicosities in the vertebral canal In a group of 181 surgically explored cases for intervertebral disk 12 patients (6 2 per cent) proved to have sciatic pain due to epidural varicosities [27] The sciatic and obturator veins branches of the hypogastric are often involved in a postphlebitic dilatation and may contribute to the so-called pelvic neuritis" which follows iliac or caval obstruction

When the obstruction is acute such as in ascending iliac vein throm bosis the unilateral or bilateral edema of the thigh extends into the buttocks and the lower part of the abdominal wall may show pitting Urinary fre quency and rectal spasm may be present If the thrombosis spreads upwards it may involve the renal vein or veins Hematuria and albuminuria are pres ent, and terminally a renal vein thrombosis is found This however is a rather infrequent complication and may be due to large periaortic lymph glands in spreading carcinoma of the bowel

Treatment of the acute phase is energetic anticoagulant therapy which will be described in detail The foot of the bed is elevated on 10 to 12 inch blocks and elastic compression is applied Sympathetic blocks are not em ployed unless there is cyanosis and vessel spasm Vena cava ligation is done only if anticoagulant therapy is contraindicated or if pulmonary embolism occurs in spite of anticoagulant therapy

The late effects of vena cava ligation are such that our clinic only does them as a life-saving measure to combat pulmonary embolism. While the postphlebitic syndrome including marked saphenous and perforator valvu lar incompetence induration and ulceration can be treated in the usual manner the persistent venous hypertension will be a permanent handicap and requires continuous elastic support

The treatment of chronic vena caval obstruction by excision or bypass of the closed segment has been attempted in several clinics, but the long term results are not known Our group has no personal experience with such a procedure

Venous Thrombosis in the Pelvis

There is considerable difference of opinion regarding the recognition and clinical significance of pelvic venous thrombosis Of course there are large perirectal periuterine periprostatic and perivesical plexus of veins and the number of phleboliths seen in pelvic roentgenograms is large (fig. 178) But while many authors ignore these as an important source of ascending thrombosis and pulmonary embolism, persuasive statistics indicate that 50 per cent of pulmonary emboli originate from above the level of the inguinal ligament,[28] and that the incidence of pulmonary embolism following prophy- lactic femoral vein ligations is as high if not higher than that in a control

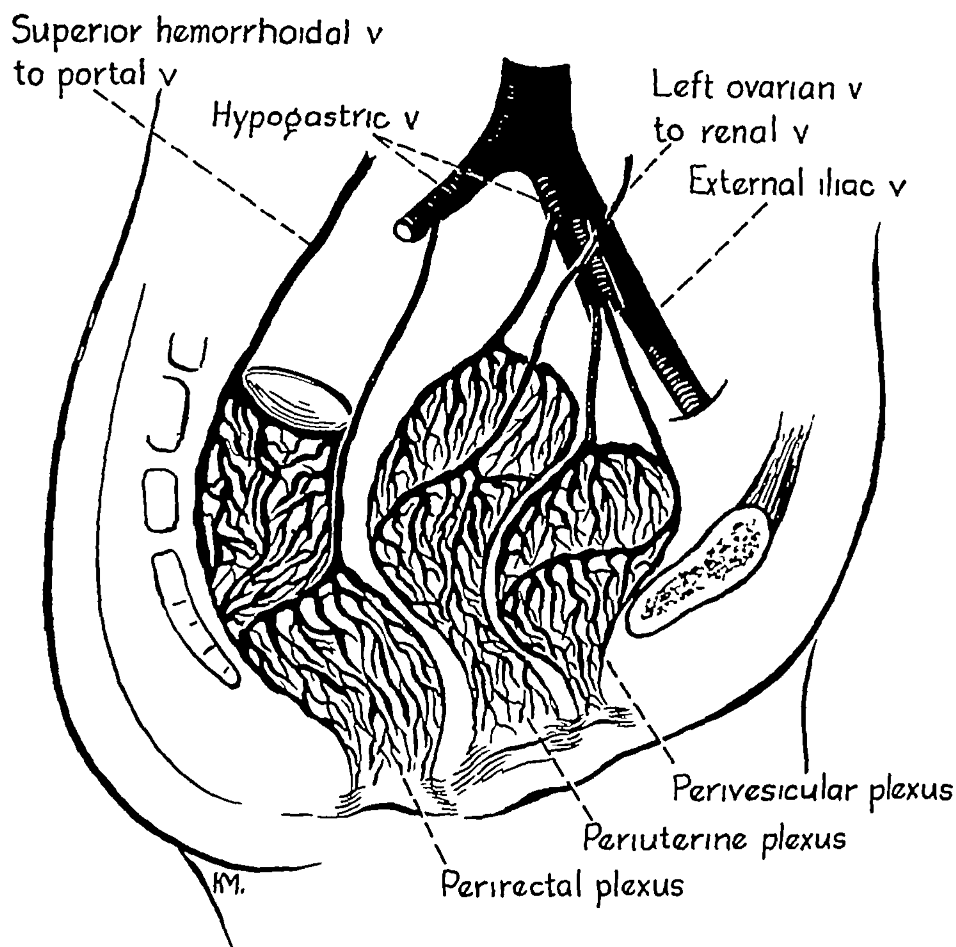


FIG 178 Pelvic venous plexus and their drainage The perirectal, periuterine and perivesicular plexus are shown with their drainage into the hypogastric vein Note the drainage to the portal and renal veins

series without ligation [29] Some of our surgeons at the University of Illinois have observed fatal and nonfatal postoperative pulmonary embolism in spite of prophylactic superficial femoral vein ligation, this emphasizes two other potent sources of emboli, namely, the veins of the adductor muscle group and the veins of the pelvis

The recognition of *pelvic venous thrombosis* is difficult, if often impossible, when the thrombi are bland and noninflammatory, *i e*, if they fit into the category of phlebothrombosis as defined by Ochsner and DeBakey [30]

It was especially the French Surgical School in Toulouse, headed by Professor Ducuing, which drew our attention to such silent or latent pelvic thrombi in a monograph published in 1929 [31] Dysuria, rectal spasms and minimal prepubic or vaginal swelling, together with elevation of temperature and tachycardia, are naturally such vague and nonspecific postoperative findings, especially after pelvic laparotomy or childbirth, that nobody can make a positive diagnosis on such a basis Of course, the palpable cord along the utero-ovarian veins may differentiate a pelvic phlebitis from an edematous infiltrated parametrium, and one occasionally gets a positive diagnosis from an obstetric or gynecologic consultant Nevertheless, the diagnosis of an acute pelvic vein thrombosis is almost never possible, unless an associ-

ated common iliac vein thrombosis is manifest In the case of a sudden pulmonary embolism without any localizing signs the pelvic veins are always suspect, together with a thrombus of cardiac origin

In chronic pelvic thrombosis phlebography has been widely used by Ducuing's pupils notably Guilhem and Baux [32] Our own limited experience with visualization of the pelvic veins by an intraosseous injection of the horizontal ramus of the os pubis or the major trochanter indicates that while interesting pictures may be obtained they hardly contribute to methods of relief The pelvic postphlebitic syndrome consists of (1) vague intermittent attacks of neuralgia along the sciatic obturator and gluteal nerves which accompany the incompetent and thus varicose branches of the hypogastric veins (2) increased edema pain and venous stasis in the postphlebitic extremity during menstrual periods and (3) pubic vaginal and gluteal varicosities which do not connect with the saphenous or femoral systems Since pelvic venous thrombosis is very frequently associated with deep venous insufficiency of the lower extremities this syndrome will be further described on page 276

Unless one can ligate or remove the incompetent hypogastric vein or its visceral branches during a pelvic laparotomy treatment of the pelvic postphlebitic syndrome is unsatisfactory Sometimes the deep visceral boring pain may be relieved by paravertebral sympathetic blocks this method deserves a trial especially in individuals with a high vasomotor reactivity

Suppurative Pelvic Thrombophlebitis

Truly a pelvic periphlebitis and lymphangitis following abortions infected childbirths or pelvic laparotomies this produces chills a septic type of temperature and an intermittent bacteremia which does not always yield a positive blood culture In a few personal cases intensive antibiotic therapy combined with massive intravenous doses of heparin has yielded excellent results, in that not a single patient was lost or developed pulmonary embolism Conrad Collins and his associates[33] reported on 62 cures out of 70 patients treated by ligation of the vena cava and both ovarian veins combined with antibiotic therapy and sympathetic blocks Their vast experience with the type of material seen at the Charity Hospital New Orleans in late cases of septic abortion demands serious consideration of this type of therapy

Iliofemoral Venous Thrombosis

It is almost certain that the concept of Aschoff is erroneous, implicating the inguinal ligament as responsible for the location of thrombosis in the iliofemoral segment [34] It is true however that at this bottleneck a number of propagating thromboses ascending from the saphenous superficial femoral and deep femoral veins and descending from the hypogastric system through the iliac segment become suddenly manifest (fig 179)

The thrombotic occlusion at the level of the groin has characteristic and

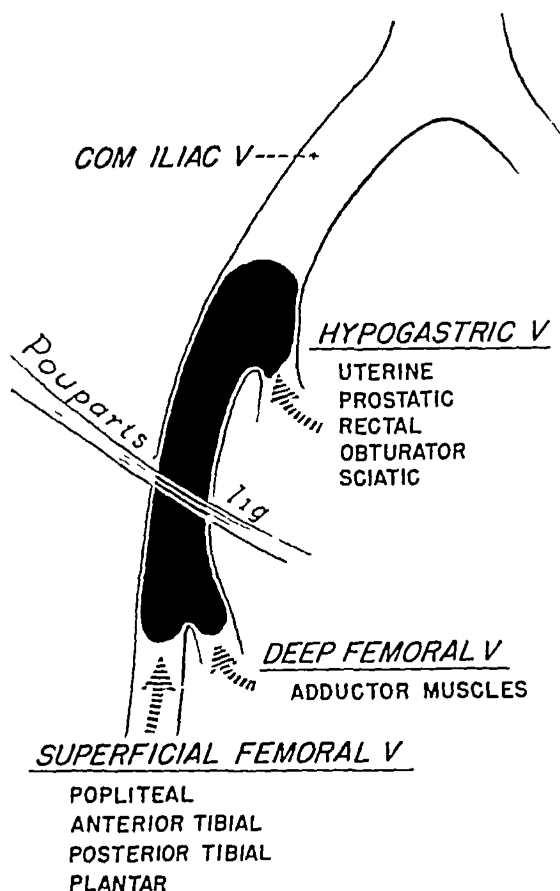


FIG 179 The bottleneck at Poupart's ligament The ascending thrombosis from the superficial and deep femoral veins and the descending thrombosis from the hypogastric system obstruct the iliofemoral segment

dramatic symptoms There is pain in the groin with tenderness on palpation and a boggy edema mostly due to enlarged lymph glands In the acute phase the edema is soft and pitting and extends to the suprapubic and lower abdominal regions and the upper thigh When there is neither edema nor tenderness below the knee, the chances are that the origin of the thrombosis is in the pelvic veins or the adductor muscles of the thigh. This, however, is rather the exception than the rule Ordinarily the patient complains of ache and tenderness to palpation in the bottom of the foot, around the ankle or in the calf *It takes several days to a week* (this is often accelerated by massage) before the thrombus reaches the groin and, as will be pointed out, much valuable time is lost during this period

A collateral network of veins makes its appearance as the edema begins to subside, but this does not always appear, indicating that either the vein has sufficiently recanalized or that deep collateral circulation has taken over

The treatment of this iliofemoral venous occlusion must be energetic in order to minimize the postphlebitic sequelae which may become permanent and disabling *Edema* is combated by high elevation of the foot of the bed on shock blocks or chairs at least 10 inches from the ground The customary elevation of the lower leg on pillows or with a break in the bed at the level of the knee does more harm than good, creating angulation and stasis at the knee or groin (fig 161) For this reason, the use of a postoperative

Trendelenburg position for the first 24 hours as first advocated by Howard K Gray has been a routine order on our service both for prophylaxis and treatment of venous thrombosis and better pharyngeal and bronchial drainage [35]

Cyanosis which occurs first to a greater or lesser degree indicates terminal venous stasis If associated with coldness and diminished peripheral pulses, it will be readily relieved by paravertebral block In our material however such vasospastic forms are rather rare constituting not more than 10 per cent of the cases the majority of patients have warm extremities with bounding pulses and higher oscillometric indices than those on the unaffected extremity They are therefore not treated by paravertebral sympathetic blocks which in any case should *always precede anticoagulant therapy and not be done on a heparinized patient* [36]

There is a rare form of extreme cyanosis following massive iliofemoral venous occlusion called *phlegmasia cerulea dolens* or *blue phlebitis* first described by Gregoire in 1938 [37]

While mild forms may exist and be readily managed by maximal elevation sympathetic block and heparinization, there is a highly acute virulent type of the disease which rapidly leads to gangrene or death In a collective review of 56 cases DeBakey and Ochsner encountered 19 deaths and 24 gangrenes [38] In this country excellent reviews together with a report of personal cases have been published by a number of authors [38] A complete bibliography and summary of a rather extensive literature may be found in the excellent monograph of Olivier [39]

The syndrome starts with extreme cyanosis pain edema and loss of arterial pulsations, and yet sympathetic block does not seem to affect it Aspiration of the iliofemoral venous segment yields jet black, thick blood not necessarily firmly clotted. One definitely gets the impression of a *massive terminal venocapillary thrombosis* A study of amputated legs does not reveal any arterial obstruction Veal and his associates[38] have emphasized maximal elevation of the limb to facilitate venous drainage While hypotension is part of the picture one wonders whether a shocklike state does not really precede the occurrence of blue phlebitis

In a recent personal experience, an aortic and iliac endarterectomy on the right was accompanied by severe hypotension on the operating table. Next day the left, uninvolved extremity showed massive edema of the thigh and a cold purple extremity to the knee. This extremity had previously been sympathectomized The femoral pulse remained vigorous, but in spite of this the extremity was lost at midthigh. While there was some vascular sclerosis, the major arterial pathways were patent. The femoral vein, when ligated at the groin prior to amputation, was thin-walled and patent in spite of the huge edema of the thigh. There were massive areas of muscle necrosis and skin and subcutaneous tissue failed to bleed at the level of amputation

Since ischemic extremities tolerate elevation very poorly one hesitates to use gravity drainage for such an extremity Yet because of the great danger of gangrene maximal elevation and possibly intravenous fibrinolysin may have to be resorted to in future cases

Venous Thrombosis of the Lower Extremities

Certain clinical forms of this condition are readily distinguished, but obviously they represent various stages or phases of a stationary recurrent or rapidly progressively lesion There are evidently acute, subacute and chronic forms of the disease, and effective treatment has to be adjusted to the stage of thrombotic disease

Sites of Venous Thrombosis

(A) SUPERFICIAL PHLEBITIS In previously nonvaricose veins, this is always suggestive of a phase of thromboangiitis obliterans I have already described in chapter 9 this manifestation of such a hyperergic vasculitis Characteristically, the dorsal veins of the foot or branches of the plantar veins are affected, but there may be short streaks of red, hot periphlebitis in the calf or even in the thigh The clinical impression is that of an ascending segmental perivenous lymphangitis, which may leave the arterial system intact A biopsy may show the characteristic foreign-body giant cells and lymphocytic infiltration (see the discussion of thromboangiitis obliterans, p 162)

Saphenous ligation, bedrest and anticoagulant therapy are frequently employed, but such treatment is useless The treatment of choice consists of (1) complete abstinence from tobacco, (2) continued ambulation with elastic compression, (3) mild parenteral protein therapy with small, subreactionary doses of typhoid vaccine or Pyromen, or (4) a series of twelve injections of sodium thiosulphate, which seems to bind or chelate the toxic or allergic substance

Very rarely, the deep veins are affected and this may give rise to pulmonary embolism Such patients may show evidence of a migrating phlebitis and this process may appear in any segment of the venous system

It is customary to include the superficial phlebitis of incipient thromboangiitis among the cases of *migrating phlebitis*, yet this entity has many other sources of origin. When confronted with red streaks or nodules of spontaneous superficial phlebitis, the following lesions have to be looked for

Cancer of hidden origin, not only of the pancreas, but lung cancer, cancer of the ovaries or any metastasizing lesion, may manifest itself in migrating phlebitis This may well be due to the thromboplastic nature of cancerous tissue, and there is experimental evidence for this [40] In a suburban hospital I saw a patient with migrating phlebitis of the arms and legs, whose ovarian carcinoma had been unrecognized Considerable work has been done on the disturbance of the clotting mechanism in patients with pancreatic disease and it has been suggested that the external secretion of the pancreas, when backed up into the bloodstream, produces increased coagulability of the blood [41] Our early studies with heparin resistance indicated that patients harboring cancer show an increased resistance to heparin Clinically, it is a frequent observation that thrombophlebitis in cancerous patients will simply

not respond to heparin therapy in fact this resistance heightens the suspicion of a hidden unrecognized cancer

"Idiopathic recurrent thrombophlebitis a term coined by Briggs in 1905 [43] is preferred by Nelson Barker for migrating phlebitis Barker has given this problem a great deal of thought and study [44] Infectious metabolic and allergic causes have all been reported as being responsible In our clinic we have seen them associated with malaria, following influenza in gout and after viral hepatitis When all obvious causes can be excluded hidden cancer, some focus of infection producing vascular sensitization, gout and polycythemia should be thought of

While the first suspicion is always centered on thromboangiitis obliterans this may not appear in the arterial tree for several years and in order to avoid generalization of the process the diagnostic acumen of the best available internists must be sought Many years ago one of our patients a rather obese young man who had intermittent attacks of superficial and later deep thrombophlebitis died suddenly of pulmonary embolism At autopsy many visceral thromboses were found Vinther Paulsen[45] has especially emphasized visceral involvement in this disease and pointed to its serious consequences Of 111 collected cases 20 per cent died Renal hepatic suprarenal superior vena caval and cerebral venous thromboses have been found among the causes of death Pulmonary embolism was also observed

While undoubtedly this is a biased sample and while many ambulatory and nonheparinized patients are never included in such statistics every effort must be made to establish an etiologic factor in order to treat it Barring the availability of a diagnosis, (1) one must ascertain whether or not there is an increased clotting tendency (p 32) and if so establish long term anticoagulant therapy (2) if a low grade septicemia is encountered, long term anticoagulant therapy with a wide spectrum oral antibiotic is useful (3) should a positive culture be obtained from some focus, autogenous vaccine therapy has helped in a few cases and (4) small subcutaneous doses of typhoid vaccine or sodium thiosulphate (described under the treatment of thromboangiitis obliterans, p 173) are very effective

Locally small 60 to 80 roentgen unit doses have been applied.[46] This method will be described in detail under treatment of superficial collateral phlebitis

(B) Superficial Collateral Phlebitis By far the largest and the most poorly understood group of superficial phlebitides are the painful red streaks and patches of phlebitis and periphlebitis which occur in collateral veins and result from earlier deep venous obstruction The literature is mostly silent on this subject and its appearance stimulates both patient and physician to unwarranted therapeutic activity Such patients either give the history or show the characteristic collateral pattern of an old deep venous occlusion (fig. 180) The flow in these veins is slow and the wall is thickened and shows low grade inflammation and fibrosis The skin over such a vein is frequently warmer and a puncture of the vein slight trauma or some systemic infection readily sets up a patch of phlebitis This may occur on the



FIG 180 The characteristic pattern of collateral venous network Note the tortuous collaterals on the thigh and in front of the tibia The patient's veins have been marked for ligation and stripping

calf, often in the anterolateral or pretibial veins, in the anterolateral veins in the thigh or in the superficial collaterals of the abdominal wall when the iliac veins or the vena cava are occluded This phlebitis may recur several times a year and yet has nothing in common with a migrating phlebitis It has a perfectly good local reason and in our experience is not emboligenic While it originates in a deep venous obstruction, there is no flare-up in that system

If the lesion is very painful and the periphlebitic exudate quite marked, a few days of bed rest with hot fomentation may be prescribed, but keeping the patient in bed for weeks and frightening him with the specter of pulmonary embolism is unncessary Equally useless is anticoagulant therapy, since there is no evidence of a deficit in the clotting mechanism and the lesion is an inflammatory periphlebitis Nor can one advocate saphenous ligation at its junction with the femoral vein, because contrary to the case of a pure saphenous vein phlebitis, the inflamed segment is fed by and connected with incompetent perforators and not much reduction in venous pressure is accomplished by this procedure

More useful is adequate elastic compression with ambulation and small doses of roentgen ray treatment, which we described in detail in 1949 [46] The dosage must be small, not exceeding 80 roentgen units, and not repeated until the mild inflammatory reaction which it produces has completely subsided In an early communication, "*Resting Infection*" *in Varicose Veins*,[47] it was pointed out that such veins are thickened and painful on

pressure, phleboliths are frequently palpable and elevated skin temperatures often 3 to 4 C higher than on a symmetrical spot on the opposite leg are not uncommon A simple venipuncture of such a vein may result in an acute patch of thrombophlebitis When roentgen rays were applied to such an area, the skin reacted with an increased rise in temperature (fig 181)

From these initial observations two therapeutic conclusions were reached (1) do not inject sclerosing solution in the collateral veins especially when they harbor infection and (2) use roentgen ray therapy for the recurrent attacks of phlebitis to cool off an acute flare up The procedure which has gradually evolved after 25 years of experiences with roentgen ray treatment of phlebitis is as follows [46] from one to six treatments of 80 r are given using 200 kv and 20 ma at a focal skin distance of 50 cm with a medium Thoreus filter * The half value layer is 1 9 cu mm An area of 400 square cm or less is treated each day

* The Thoreus filter is a primary filter of tin, together with a secondary filter of copper In the 200 to 400 kv range, such a filter selectively transmits relatively more of the short wave length part of the beam than does a filter consisting of tin or copper only (Glossary of Radiology and Atomic Energy National Research Council, Washington, D C., 1949)

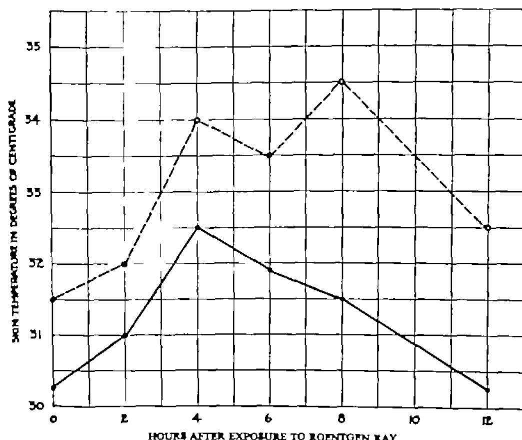


FIG 181 Skin temperatures of normal skin and skin overlying a collateral vein before and for 12 hours after a single dose of 125 r units. Note that the affected side is slightly warmer before radiation. After application of 125 r units with 2 mm aluminum filter the affected side showed an exaggerated thermal response. The straight line is the temperature of the control side the interrupted line is where "resting infection" was present. (de Takats, G Am. J M. Sc., *194* 57 1932.)

It is important to wait for the subsidence of inflammatory response, which may take from three to seven days. The thrombus is not apt to be lysed but the periphlebitic exudate containing radiosensitive lymphocytes rapidly responds in about 85 per cent of the cases Relief of pain is often dramatic and the patients remain ambulatory with adequate elastic compression The type of deep thrombophlebitis benefiting from roentgen therapy will be discussed on page 290

The ultimate, definitive treatment of these recurrent postphlebitic varicosities is an extensive ligating and stripping procedure after all possible sources of focal infection have been eliminated and when the process is quiescent

(C) Acute Superficial Phlebitis in Saphenous Varicosities. The previously described collateral phlebitides are often mistaken for this lesion, yet its significance and treatment are different Valvular incompetence of the long or short saphenous system is not frequent to start with, and inflammation or thrombosis are comparatively rare These will be brought on, however, by direct trauma, by infected teeth, tonsils or cervix or by enterocolitis, while they may start as a small patch, the entire column of blood in the long saphenous vein may rapidly clot, ascending to the groin Such an event may follow a sclerosing injection around the distal end of the vein and close up the entire segment, but with considerable periphlebitic reaction, however, there is no involvement of the perforator or deep venous system There is no edema except that which localizes as a tubular tender mass around the involved vein

In such a situation, a proximal vein ligation at the saphenofemoral junction is a simple and logical procedure In the first place, the clot may ascend into the femoral vein, especially if the saphenous bulb is large and can be readily aspirated at the time of dividing the saphenous vein Second, in relieving the back pressure prevailing in the valveless saphenous trunk, pain in the erect position is relieved There is, again, no need for prolonged bedrest, for anticoagulants or for paravertebral blocks The patients can be ambulatory with elastic compression, and roentgen ray therapy may be used if the periphlebitis is slow in subsiding Frequently, the vein recanalizes and then a formal stripping procedure can be done in the "cold state"

(D) Deep Venous Thrombosis There are venous plexus in the sole of the foot, in the calf muscles and in the adductor muscles, which form stagnant pools and serve as a nidus for a spreading thrombus (fig 182) Small thrombi are often silent and become manifest only when protective muscle spasm, small collateral edema or a vague diffuse "visceral" type of pain appears

Plantar vein thrombosis[48] There is numbness and cramping in the bottom of the foot and pain on pressure over the lateral surface of the foot A little later the posterior tibial vein may become tender between the inner malleolus and the Achilles tendon, with a slight pitting edema filling out the hollow area at the medial malleolus The process may stop here and result in atypical patterns of varicosities coursing over the Achilles tendon

from incompetent perforators connecting the deep veins with these collaterals.

The plantar vein thrombosis, however may ascend to the deep veins of the calf or calf muscle vein thrombosis may result from primary involvement of this plexus Here the muscles are tender to pressure with a doughy consistency and a small elevation of skin temperature Dorsiflexion of the foot causes pain in the spastic muscles as described by Homans [49] but this sign is negative in about 40 per cent of the cases it may be positive when the cause of muscle spasm is revealed to be due to root pain from compression at vertebral level Edema will not develop until a number of the deep venous channels including the popliteal vein become involved Lowenberg s use of a blood pressure cuff pumped up in 10 to 15 seconds to a point where it causes pain on the affected side is a helpful quantitative test for pressure pain While the normal calf will stand a pressure of 160 to 180 mm of mercury without pain the calf harboring thrombosis may become painful at 60 to 100 mm of mercury [50]

There are other methods aimed at accentuating a minimal or subclinical venous stasis A pressure of 60 mm of mercury applied with a cuff to the thigh may provoke pain and cramp in the calf muscles The oscillometric

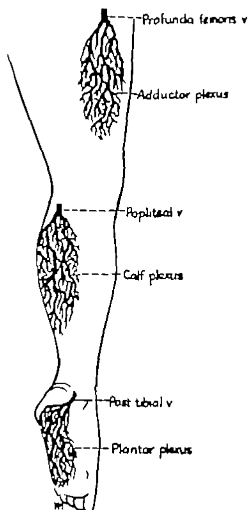


FIG 182. The plantar calf muscle and adductor muscle plexus of veins which drain into the posterior tibial, the popliteal and the deep femoral veins, respectively

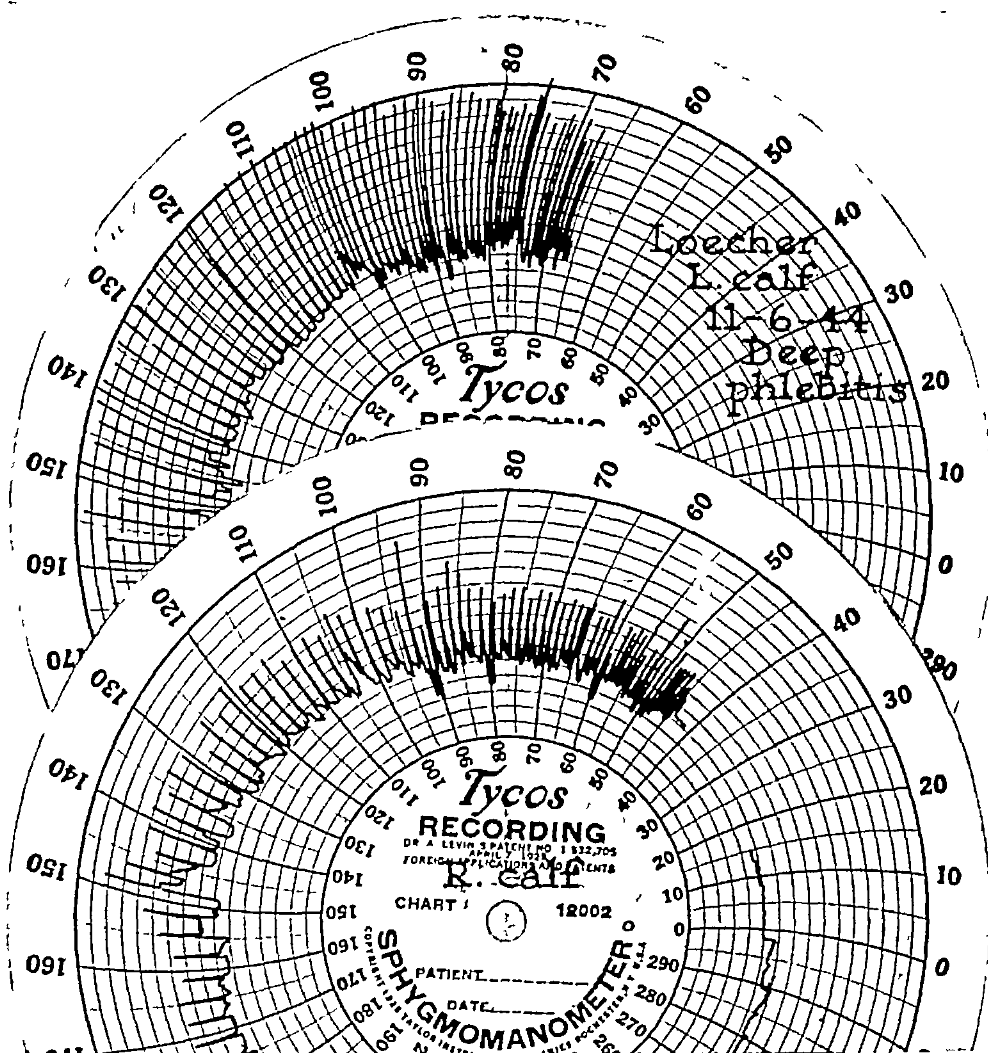


FIG 183 Oscillometric curves obtained by a Tycos recording sphygmomanometer The inflammatory reaction and the obstruction to venous outflow are responsible for the higher oscillations on the affected side. This is a visual demonstration of the "throbbing" complained of in the early phases of deep phlebitis

curve, even at an early phase, is higher on the affected than on the control side (fig 183)

These early signs and symptoms of calf muscle vein thrombosis are important for two reasons When they are positive, early intensive therapy can be instituted, preventing late sequelae When they are absent, much anxiety, bedrest and long term anticoagulant therapy can be eliminated

Adductor muscle vein thrombosis This is an infrequent but important site of thrombosis, since the venous plexus drains into the profunda and hence into the common femoral vein, and is not excluded from the systemic circulation by ligation of the superficial femoral vein It is characterized by muscle spasm, tenderness and edema in the adductor area medially and below Poupart's ligament There is no swelling or venous distention of the ankle, calf or lower thigh It sometimes accompanies an iliac or common femoral venous thrombosis by retrograde spread into this area

Iliofemoral venous thrombosis The obstruction at this level, as shown in figure 179, leads to massive edema of the foot, lower leg and

thigh, this is first soft pitting and cyanotic, but later becomes hard with an added lymphatic component, white and nonpitting As shown with Zimmer man in our early experimental studies,[51] massive thrombophlebitic edema can be produced in the dog by injecting a concentrated tissue extract into the femoral vein and creating a hemorrhagic, fibrinous exudate in the tissues with a high protein content Secondary lymph stasis seems an important factor and the anoxic capillary stasis leads to endothelial permeability

The *pain* which is often severe and gnaws at the groin and another deep constricting ache over the entire extremity are due to two different mecha nisms The inguinal pain is almost invariably due to the enlargement and inflammatory reaction of the inguinal lymph glands Homans and Zollinger[52] have emphasized the lymphatic component in the production of phlegmasia alba dolens the milk leg The severe constricting ache often accompanied by cyanosis is due to vasoconstriction which is present at the onset and may not be apparent when the patient is seen a day or two later Together with cyanosis one may encounter a cold pulseless foot simulating arterial obstruction

The late consequences of iliofemoral venous thrombosis will be dis cussed under the postphlebitic syndrome page 281 It will suffice to say here that every effort should be made in the acute phase to (1) eliminate or mini mize edema, (2) abolish vasoconstriction and (3) stop the propagation of thrombosis both distally and proximally by anticoagulants or proximal venous interruption

Stages of Thrombotic State

A *subclinical phase* of the thrombotic state exists which is either symptomless or is accompanied by such vague and nonspecific symptoms that a positive diagnosis is impossible I have already referred to the mild ache around the ankle or in the calf which may or may not be substantiated by objective findings a suspected thrombosis which may be treated by anti coagulants, since it can give rise to pulmonary embolism without ever be coming manifest

After such a latent or suspect phase however definite localizing symptoms may occur and, as will be pointed out under treatment (p 339) in tensive anticoagulant therapy is our method of choice In the *acute phase* depending on the extent and site of the thrombosis local inflammatory and systemic reaction may be present Aschoff in his *Lectures on Pathology*[34] spoke of a bland static thrombus which he attributed to stasis and an inflammatory" type characterized by much perivenous reaction with pain and fever Ochsner and DeBakey[30] coined the terms of phlebothrombosis and thrombophlebitis and these diagnoses are clinically useful since they emphasize the different aspects of the disease However as we have always maintained the same patient may have a bland, noninflammatory deep venous thrombosis in the calf which becomes a painful inflammatory thrombophlebitis when it reaches the groin and affects the lymphatics and

lymph glands Frequently one sees patients who are diagnosed as having thrombophlebitis in one leg (usually the superficial collateral type of phlebitis) and phlebothrombosis in the other leg, manifested by an edema of the calf due to thrombosis of the deep veins

Actually the intravascular clot may produce little or a great deal of perivenous reaction, and the latter, when it involves the perivenous lymphatics and nerve endings, is responsible for the *periphlebitis* Such periphlebitis is especially favored by a pre-existing low-grade infection in the lymphatics which exists in patients with fungus infections between the toes or in the vagina The term *phlebodynia* was coined by I S Wright, this was reported from a nurses' dormitory in Huntington, West Virginia, where an epidemic of periphlebitis broke out along the course of the saphenous vein in 19 student nurses, in the form of pain, redness and swelling along the long saphenous vein The lesion was in the wall of the vein and no thrombus was present [53] Through the courtesy of Dr Pearson, we had the opportunity to examine one of these students, and we feel that this was a case of retrograde lymphatic spread from a vaginal fungus Such patients may develop a mild persistent edema because of the lymphatic block in the groin and this may be an early beginning of an infectious lymphedema (see chapter 14) It has been thought to be not unlike the phlebitis in thromboangiitis obliterans [18]

The recognition of this infectious factor which, of course, may come from the toes, from the vagina or from the pelvic organs, is important since its elimination may be the best prevention against recurrence In addition to antibiotics, bacterial or fungal allergy should be considered and, as pointed out in chapter 14, autogenous vaccine, if available, is very useful

When the acute phase of thrombosis subsides, there remain sequelae anywhere from a mild periphlebitic scarring and cutaneous pigmentation to large patches of induration surrounding the inflamed vein, to hard plaques of indurated and necrotic subcutaneous fat and to massive resistant postphlebitic edema, in which lymph stasis is a big factor. The postphlebitic syndrome is such an important clinical entity that its management will be described in detail

Any time from the vague, subclinical onset of thrombosis, throughout its acute course and in a recurrent phase, sometimes 8 to 15 years after the acute onset, pulmonary embolism may develop This entity will receive separate treatment Its prevention lies in the early intensive management of peripheral venous thrombosis, although—and this needs to be stressed—50 to 75 per cent of pulmonary embolic occur without any warning at the periphery, and a certain percentage come from the right side of the heart

Treatment of Venous Thrombosis

Reference has been made in the foregoing description of clinical entities to various forms of management Here a brief summary will be given of the available measures, emphasizing the procedures employed in our clinic

ANTICOAGULANT THERAPY The surgeon s anticoagulant is heparin There may be situations in which long term anticoagulant therapy with oral prothrombin depressants is indicated Early experience with Dicumarol however taught us that its control with prothrombin levels is difficult and that, most of the time, token levels or at least unprotective levels are administered [54] As far as heparin substitutes are concerned, our laboratory has studied two of these large molecule polysaccharides notably Treburon and dextran sulfate * Aside from platelet depression and alopecia as frequent side effects, the greatest disadvantage of these drugs is that they cannot be administered subcutaneously in concentrated solution Actually heparin is now our anticoagulant of choice the fact that oral or sublingual administration is ineffective makes it all the more efficient since as will be pointed out, rapid and large concentration in the blood is desirable at least during the acute phase of thrombosis

HEPARIN THERAPY The proper administration of heparin is based on the recognition that each patient has his own heparin tolerance[42] and that even in the same patient the response to heparin varies according to the stage of the thromboembolic disease Thus in an acute phase of the disease in which there is pain edema, inflammatory reaction in the lymphatics, fever leucocytosis eosinopenia increased sedimentation rate and a shortening of the clotting time, far more heparin is required to lengthen the clotting time than if the thrombotic process is subsiding, either by sudden defervescence diuresis and disappearance of edema or by a slow gradual improvement which frequently results in a chronic recurrent phase of the disease The two types of convalescence by crisis or by lysis are quite distinct,[55] and so are certain signs of the alarm reaction in acute massive thrombosis, such as eosinopenia and water and sodium retention

To illustrate the two types of convalescence the following cases are presented.

(1) M.K. entered the hospital with a huge acute thrombophlebitic edema, an unrecognized pulmonary embolism and uncontrolled diabetes (fig. 184) He received 5,200 mg. of heparin during the first week, sometimes as much as 1 000 mg. a day The thromboembolic disease subsided by crisis on the seventh day manifested by sudden diuresis, a sharp rise in clotting time and increased sensitivity to heparin. An easier and more satisfactory control was established in the case of M W age 22, whose thrombosis only reached to the knee and who was seen 24 hours after its onset. She was a slight, anemic girl treatment was started with three intravenous injections of 100 mg. of heparin for three days, followed by increasing doses of intramuscular heparin stabilized at 150 mg. a day which was gradually decreased and stopped on the fourteenth day (fig. 185)

(2) In contrast to such rapid convalescences, the case of Dr E.E., a 56 year old surgeon, is presented. Because of a microscopic hematuria dating from a glomerulonephritis in childhood and a preference for oral Dicumarol by the medical department, this patient illustrates a protracted convalescence. He first entered the hospital with a hard, swollen, painful left calf occurring four days before admission. He was promptly placed on 50 mg. of heparin administered intravenously three times daily and this was then increased to 100 mg. administered intravenously for a week. He was discharged with two daily doses of 125 mg. of heparin. The acute phase rapidly disappeared, the edema and tenderness were relieved, and against advice the patient stopped the heparin while at home for one week.

* Unpublished observations.

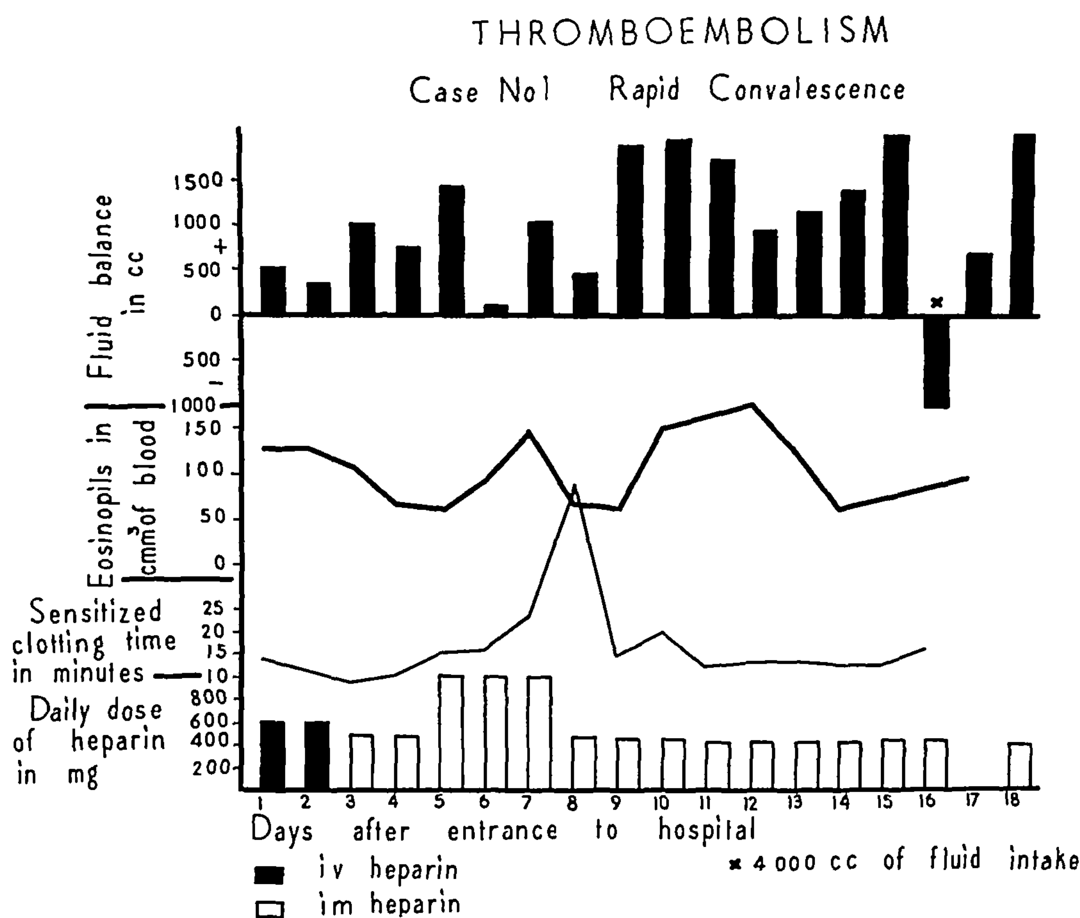


FIG 184 M K, a 63 year old schoolteacher, had massive acute iliofemoral thrombosis with compensated heart disease and uncontrolled diabetes Treatment started with 600 mg of intravenous heparin daily for two days, which did not raise his clotting time Intramuscular heparin was also inadequate, until 1,000 mg were given daily This led on the seventh day to a rapid rise in coagulation time and marked diuresis The dose of heparin was promptly cut to 500 mg daily and maintained for 18 days Re-examination seven months later revealed no edema with elastic support and no recurrence (de Takats, G Anticoagulant Therapy Surgery, *34* 985, 1953)

Five days later a right lower lobe pulmonary embolus occurred, for which he was treated by bedrest and resumption of his previous maintenance dose, *i e*, 250 mg of heparin While on this medication at home, the patient developed a second pulmonary infarct, this time to the left lower lobe The left calf became turgid again The medical department now started him on Dicumarol, maintaining prothrombin activity between 40 and 25 per cent which was kept up for three months He finally made a full recovery and has remained fully active for the last five years, with some bouts of edema His fluid balance is shown in figure 186, while on his second admission This is a protracted or "failed" convalescence, exhibiting a slow rise of eosinophils and a negative fluid balance, markedly accentuated by a 4,000 cc of fluid intake

These histories emphasize important but seldom observed rules in heparin therapy, *i e*, the dosage has to fit the severity and duration of the disease, and premature stoppage of the drug may result in exacerbation and spread of thromboembolism This withdrawal phenomenon will be alluded to again, often one is confronted with patients whose disease has been protracted by intermittent and insufficient control and whose natural defense against the clotting phenomena has been seriously impaired Recently, a group of cases was reported in which emboli occurred during heparin ther-

apy [56] None of these patients had intravenous heparin at the start and they may have had insufficient heparin absorption through subcutaneous administration

Methods of administration A *continuous drip* using 100 mg. of heparin in 500 cc of physiologic saline solution in 5 per cent dextrose or blood is used in the following ways (1) intra arterially through a polythene tube distal to a clamped artery when endarterectomy or vessel replacement is performed [57] (2) intravenously through a cut down at the ankle when an acute iliofemoral venous thrombosis is treated by regional heparinization and (3) in an arm vein in the presence of a rapidly spreading, malignant form of venous thrombosis with multiple emboli This last condition is usually encountered in diffuse spreading carcinomatosis and is regarded as an exhaustion of the clotting mechanism characterized by great resistance to customary doses of heparin The control of dosage by clotting times will be presently described

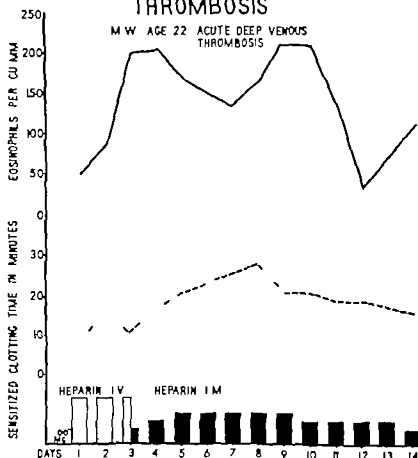


FIG. 185 Heparin therapy of acute deep venous thrombosis. After three days of 300 mg. of intravenous heparin, intramuscular heparin was continued in 100 to 150 mg. doses for 14 days. Note the rise and fall of the eosinophil count as in any other form of stress. The heparin-retarded clotting time (see text) has been kept between 16 and 20 minutes, just slightly above the upper limit of normal. (de Takats, G Management of Venous Thrombosis in Lower Extremities. Surgery 37 507 1955)

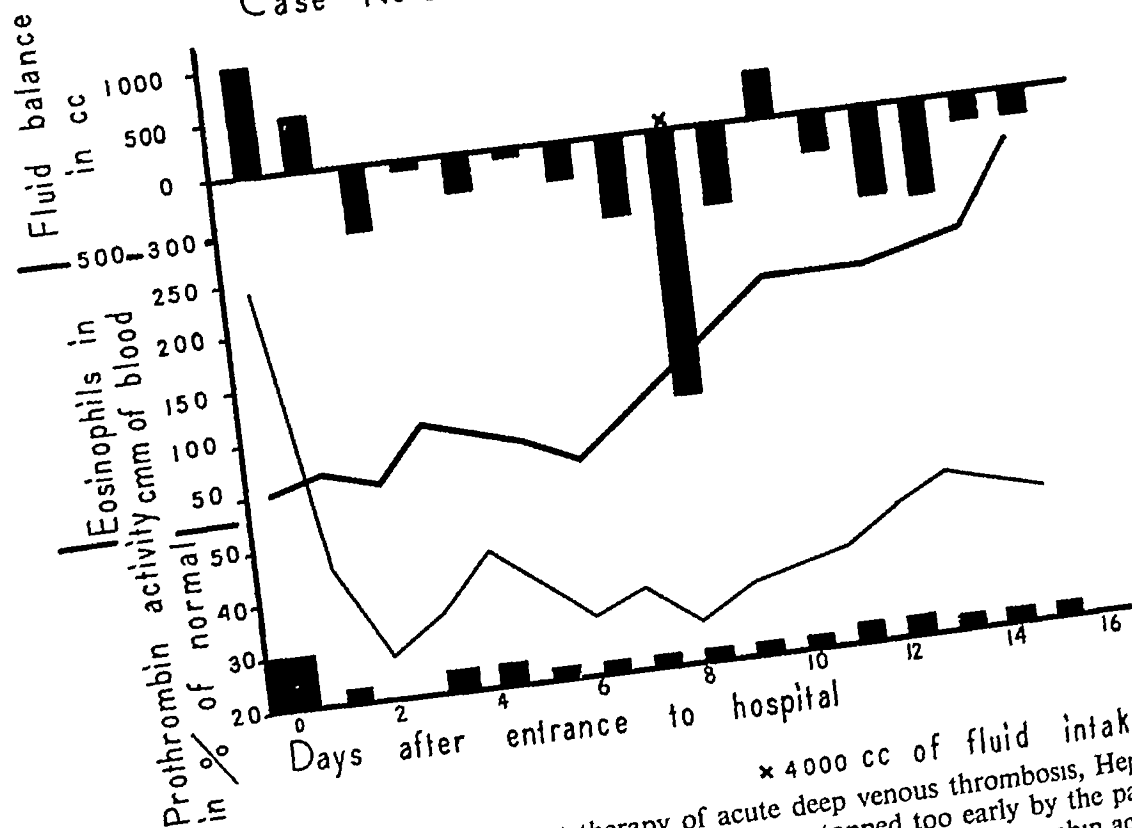


FIG 186 Insufficient anticoagulant therapy of acute deep venous thrombosis, Heparin administration was too low on the first admission and was stopped too early by the patient himself On the second admission, following two pulmonary emboli, the prothrombin activity shows good response to Dicumarol, although it was only twice below 30 per cent of normal There is a negative water balance, accentuated at the asterisk by a water load of 4,000 cc The eosinophils climb back to normal slowly The calf remained tender and swollen for many months

The *intermittent intravenous injection* of 100 to 150 mg of heparin in 1 per cent solution is given four to six times a day in the acute stage of venous thrombosis, thus an administration of from 600 to 900 mg of heparin for a few days This high dose should only be used in the acute form to arrest and revert the process to a second stage when the patient's own resistance can come into play When the fever, rapid pulse and edema suddenly subside and when the daily eosinophil count rises, the dosage is sharply cut to 200 to 300 mg. of heparin in 10 per cent aqueous solution given in two to three *deep subcutaneous injections*, these are kept up by the nursing staff, or in suitable cases by the patient himself, for a period of two to three weeks While control with clotting times is obligatory during the large intravenous doses and for a few days after the subcutaneous doses are given, the clotting times may be omitted after they become stabilized; then the patient may become ambulatory in or out of the hospital

We have abandoned the use of intramuscular heparin in a retarding medium[9] because it is painful and expensive and the plateau type of clotting time curves seems no more protective than the sharp peaks obtained with

intermittent intravenous or subcutaneous injections in aqueous solution One must fully agree with Bauer and his associates[58] that a high concentration of heparin is important in influencing the recent loose clot, and hence we do not advocate subcutaneous heparin in the early acute phase of the disease In fact, it may increase the hazard of embolism

The laboratory control of heparin therapy Heparin is far more than an anticoagulant but its action in thromboembolic disease can be best measured by clotting times of the blood In order for daily serial clotting times to be practicable on a general surgical service one must postulate that the methods be simple readily reproducible and possibly be run and reported at the bedside so as to direct the next dose of heparin Two such methods have been used *Capillary coagulation times* are simple need no venipuncture and while very crude give a good estimate of the patient's response to heparin, which notoriously varies not only in different patients but in the same patient in different stages of postoperative convalescence[42] If no other method is available this is a rapid bedside test 8 to 12 minutes of capillary coagulation time is a safe desirable level When intermittent administration is used these clotting times should be back to the preinjection level of three to four minutes before the next dose is given During the first two days of therapy it is best to have at least two capillary coagulation times determined just before the next dose, so that a stepladder type of curve can be avoided

Far more preferable however is the use of venous blood Each 1 cc of blood is mixed with 1 μg of heparin in physiologic saline solution Such tubes can be prepared in advance by the laboratory and kept in the icebox Such a *heparin-retarded clotting time* has two great advantages over the commonly used single tube or multiple tube venous coagulation times First it neutralizes the varying amounts of tissue juice which is unavoidably aspirated by venipuncture, and thus will give a slightly retarded clotting time The normal range is between 11 and 13 minutes with a small scatter above and below this level[6] (fig. 187) Second this clotting time is truly sensitized by heparin in that it will respond to anxiety premedication hemorrhage shock and surgical trauma with predictable fluctuations[7] Such a postoperative clotting curve made up of daily determinations may even direct prophylactic administration of heparin

The desirable clotting time determined by this method is between 16 and 20 minutes, and it may require massive doses of heparin up to 1 000 mg. daily during the acute phase to obtain this It has not been our aim to double or treble the normal level during anticoagulant therapy or prophylaxis but simply to restore the disturbed ratio of coagulant versus anticoagulant factors and push it slightly toward the anticoagulant side Should the heparin retarded clotting time be unavailable single or multiple tube venous clotting times can be used The desirable level is that slightly above the upper limit of normal

Heparin sensitivity A small group of patients, whether they have had heparin before or not, show evidence of drug sensitivity, such as flushing of the face bronchospasm nausea and vomiting or diarrhea An intravenous

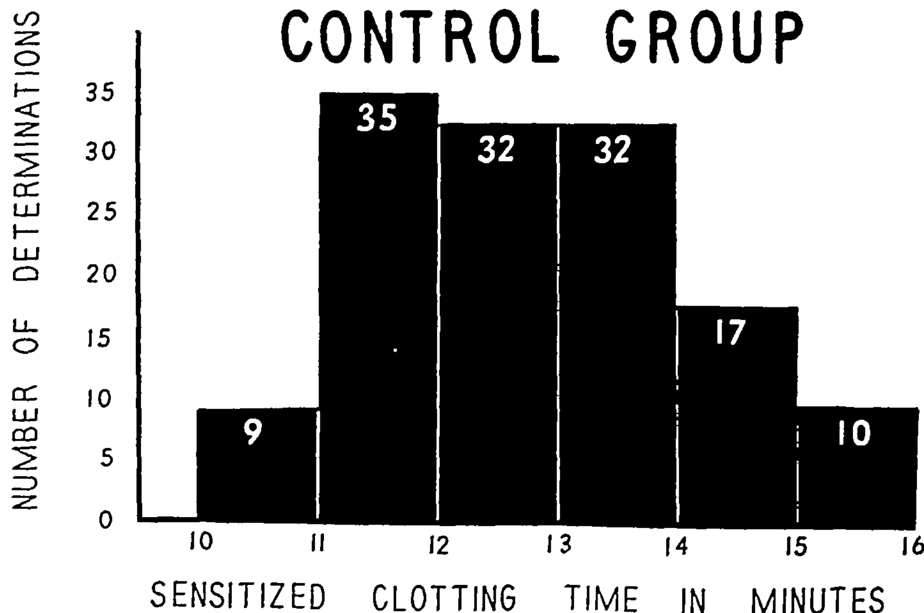


FIG 187 Distribution curve of 135 determinations of sensitized (heparin-retarded) clotting times 73 per cent of the clotting times fall between 11 and 13 minutes 1μg of heparin is added to 1 cc of venous blood (de Takats, G and Voigt, M T Response of Clotting Mechanism to ACTH Angiology, *4* 283, 1953)

test dose of a few drops to 1 cc of heparin can detect these patients. Epinephrine is a good antidote should an alarming reaction develop after an initial large dose The rate of injection should always be slow to avoid an unsuspected reaction In a study of 256 patients receiving heparin, 8 per cent showed some degree of drug sensitivity.[54]

Hemorrhage due to heparin Hemorrhage due to heparin may be encountered, due to a cumulative effect when more heparin is being given daily than is degraded or excreted Thus, on a prophylactic dose of 200 mg of heparin, the clotting curve may slowly rise, thus giving sufficient warning to reduce the dose Another cause of hemorrhage is the failure to cut the massive doses which were advocated in the early, acute phase of thrombosis, when the patient's sensitivity to heparin returns But the most frequent cause of hemorrhage after heparin occurs in postoperative patients receiving heparin for prophylaxis Here the patient's sensitivity to heparin normally rises around the third or fourth postoperative day, in the phase of defense Together with defervescence and the rise of eosinophils, the patient's response to heparin is increased Therefore, greatest care should be exerted during postoperative convalescence

Should hemorrhage still occur in spite of all precautions, protamine sulfate is given intravenously, 1 5 mg to each 1 mg of heparin being entirely suppressive For practical purposes, an injection of 100 mg of protamine sulfate given intravenously every four hours until bleeding ceases is highly effective. It may fail, however, when a massive hemorrhage has brought about a hemorrhagic state independent of heparinemia and presumably due to a shocklike state in which fibrinolysis and low platelet count appear

Blood transfusions are of course, our best standby but they may again increase hemorrhage unless the blood is carefully cross matched

Although heparin is used extensively on our service in 100 to 150 patients a year only one massive hemorrhage was encountered in the last few years and this was due to overdosage combined with a paravertebral block A huge retroperitoneal hematoma was later followed by cord symptoms [12]

Heparin rebound phenomenon Clinical observations indicate a period of hypercoagulability of the blood when heparin is abruptly or prematurely stopped Recurrent thrombosis or pulmonary emboli have been seen to occur when the active stage of thrombosis has not subsided or when insufficient initial doses have failed to throttle the acute first phase Cate[59] has given an account of the previous literature and supplied clearcut animal experiments and clinical observations to show that a significant drop in sensitized clotting times occurs 48 hours after the last injection of heparin

PROXIMAL VEIN LIGATION The principle has been adopted that a deep vein should never be ligated or divided for prophylaxis or treatment of thromboembolic disease unless the patient cannot be given heparin, or unless, in spite of adequate anticoagulant management thromboembolic phenomena continue A deep vein is an important afferent channel, and even if it does frequently recanalize after an old thrombosis leading to incompetent valves, regurgitation of blood on standing is still preferable to obstruction of a main draining channel on exercise [60]

Pulmonary embolism occurs in a large percentage of cases in well over half of them it occurs without preliminary warning and with no obvious lead as to its origin To assume that 90 to 95 per cent of the thrombi originate from the veins of the lower extremities as frequently stated in the literature is not justified by our experience In a study of 97 fatal pulmonary emboli verified by autopsy Fowler and Bollinger[61] found that the heart was the site of origin in 52 8 per cent of all cases of the remaining 47.2 per cent of fatal pulmonary emboli originating from venous thrombosis, over one half the thrombi were from the pelvis and possibly *one-third had originated in the lower extremities* For this reason we have always used proximal vein ligation most sparingly This is not to say that ligation of deep veins in thromboembolic disease should never be done The *common femoral vein* is ligated prophylactically prior to all major amputations because of the considerable incidence of pulmonary embolism in the arteriosclerotic group [31] and because the stump responds to anticoagulant therapy with hematoma and subsequent fibrosis The vena cava is ligated in patients with iliofemoral thrombosis, especially when it is bilateral when a pulmonary embolus has already occurred or in the presence of multiple emboli which anticoagulant therapy is unable to control Subsequent emboli carry a higher mortality than the first, and here the operation is life saving. In pelvic thrombophlebitis with recurrent emboli the ovarian veins are ligated in addition to the vena cava [33]

Both of these operations leave residual edema and other sequelae of increased deep venous pressure for these reasons they are employed with

strict indication While blood dyscrasias contraindicate heparin therapy, renal and hepatic impairment simply call for decreased dosage and do not call for vein ligation There is no indication for prophylactic or therapeutic division of the *superficial femoral vein*, since this does not prevent embolism in the area served by the produnda or pelvic veins and may even increase its incidence [29] It is true that, performed for prophylaxis, division of this vein produces no residual edema, but it also offers no protection and we cannot see any justification for its use

Paravertebral Sympathetic Block In the treatment of deep venous thrombosis, paravertebral sympathetic block has a definite although rather restricted role In our service we have limited its use, (1) to patients who exhibit marked vasoconstrictor activity in the affected limb, manifested by a cold, damp cyanotic extremity with decreased or absent pulses and soscilometric curves Most patients exhibit a hot, pale limb with increased ocillaltions, and in these I can see no reason for sympathetic block (2) We have also limited its use to patients who complain of a severe, deep, constricting type of pain, a "visceral" nonlocalizable pain which responds well to the interruption of sympathetic impulses. While this pain may be present in the acute phase of deep venous thrombosis, it is also encountered as a part of the postphlebitic syndrome (p 348) when it acts like a minor causalgic state and may be effectively relieved by one or several sympathetic blocks

Enzyme Therapy. Curiously enough, the lay public has long inquired about, nay demanded, a dissolution of the clot When heparin first came into use, everyone connected with its early development[62] hastened to say that heparin can only prevent propagation of thrombosis, but cannot dissolve the clot itself

Since, however, clots are dissolved in the human body by fibrinolytic action,[1] the first question which arose was whether or not heparin enhances thrombolysis by facilitating the normal fibrinolytic process While recanalization of both venous and arterial thrombi definitely occurs, *rapid patency* of a vessel occluded by a thrombus can be observed both experimentally and clinically after administration of heparin [63] In addition to observing rapid canalization of venous thrombi when regional heparinization is used, I have published case histories of arterial embolism in which intra-arterial injections of heparin given proximal to the site of the embolus resulted in the restoration of the peripheral pulse [64] However, there is always the possibility that this recanalization is spontaneous, since a reappearance of pulses is seen, especially following emboli. The possible mechanisms of such a restoration or patency will be discussed under acute arterial occlusions (p. 309). Some authors, notably Halse,[63] have strongly believed that heparin is fibrinolytic, especially when it encounters the early, soft jelly-like clot Most workers believe, however, that the fibrinolytic-antifibrinolytic mechanism is independent of heparin action and they have been more interested in activating fibrinolysin (plasmin)

Fibrinolysin is activated by a number of stimuli or by depression of

antifibrinolysin activity Several methods tried in our laboratory* failed to give consistent base lines However it is known that after transfusions prolonged ether anesthesia, and some antigen antibody reactions massive hemorrhage may occur caused by acute fibrinolysis [65] The question naturally arises as to why these physiologic responses could not be harnessed to counteract or prevent blood clotting.

The first of these attempts was made by Innerfield and his associates by using intravenous and later intramuscular trypsin [66] In experimental studies the effect of trypsin on dissolving blood clots has not been substantiated [67] In our own observations 100 000 to 150 000 units of trypsin dissolved in 500 cc. of physiologic saline solution and given intravenously once or twice a day frequently produced chills and fever Phlebitic edema was favorably influenced but there was no detectable change in the clotting mechanism. Unquestionably the degradation products of protein particularly those of fibrin may give rise to a series of enzyme reactions but this seems a round about way to activate fibrinolysis

At present, there is a widespread use of Parenzyme, an intramuscular trypsin or chymotrypsin for the treatment of thrombophlebitis Its advocates now emphasize an anti inflammatory effect which of course could be much more easily obtained by foreign protein therapy notably with Piromen The treatment of thromboangiitis obliterans with small doses of typhoid vaccine has already been described (p 174) A far more direct approach is to activate fibrinolysin (plasmin) by minute amounts of thromboplastin or trypsin Clifton[68] has reported using activated fibrinolysin to disssolve clots in vivo

Our own laboratory has experimented with aged plasma activated by minute amounts of trypsin, and has injected this material intra arterially proximal to acute arterial thromboses The material has not been used in veins Our present attitude is that while active fibrinolysis may indeed be desirable to hasten the thrombolysis the patient is being deprived of the benefits of an early administration of heparin The limitations and hazards of this method are unknown and hence its clinical use awaits further trial At present an active fibrinolysin, Actase is being given a clinical trial on our service A recent symposium on this subject contains many promising data [69]

Auxiliary Methods of Treatment Anything that will drain venous edema will decrease the postphlebitic fibrosis due to clotted plasma, proliferating fibroblasts and secondary lymphatic obstruction Elevation of the affected extremity well above heart level is useful. This must be done by raising the foot of the bed at least 10 inches or by hanging the arm down from a Balkan frame A few pillows under the knee simply angulate the popliteal and iliac veins and hinder venous return Many years ago we used wooden splints and feet cradles but these were abandoned. If the thrombophlebitic leg is very painful due to a protective muscle splint, hot fomentations are useful but can ordinarily be stopped in four to five days In 1933 I

* Unpublished observations of G de Takats and M T Voigt, and unpublished observations of G de Takats, T D Thompson and V Zaratian.

described a combination of a low-salt diet, ammonium bromide and intravenous mercurial diuresis in the management of acute thrombophlebitic edema [70] Both experimentally[71] and clinically we found a more rapid disappearance of acute thrombophlebitic edema with this method of dehydration

There is, however, a serious objection to this rapid diuresis Such patients harbor an acute thrombosis and their clotting mechanism has undoubtedly shifted to the coagulant side (part I, p 31) Sudden excessive diuresis favors thrombosis and increases heparin resistance Rapid dehydration accomplished with digitalis and Mercuzanthin, with loss of 42 pounds in two weeks, renders the patient resistant to heparin,[72] there are sufficient numbers of cases known where thromboemboli occur after sudden fluid loss

For this reason, the intense use of diuretics has been abandoned in the treatment of acute postphlebitic edema That is not to say, however, that the newer, oral type of diuretics, especially Diamox, cannot be used This drug in 250 mg daily doses eliminates fluid retention in an extremity, even in a chronic phase, and is useful as a preoperative preparation in the postphlebitic syndrome Diuril in 500 mg doses has also been used

The use of the roentgen ray in the treatment of superficial phlebitis has already been discussed (p 275) [46] In calf muscle or adductor vein thrombosis we have not employed it However, in deep venous thrombosis, which is invariably accompanied by tender, hyperplastic inguinal popliteal or axillary lymph glands, small doses (60 to 80 r units) of roentgen ray directed toward the enlarged lymph glands will quickly reduce pain and may well clear the sinusoids of fibrinous debris In fact, it has been proposed long ago that roentgen ray liberates proteolytic enzymes and gamma-globulin from lymphocytes,[73] and this is a form of enzyme therapy Roentgen ray also degranulates mast cells and may thus liberate heparin [6a]

2 Pulmonary Embolism

The true incidence of this sudden insidious complication of peripheral venous or intracardiac thrombosis is not easy to estimate Numerous statistics are available concerning its occurrence after various types of operations, after childbirth, in cardiac patients or following trauma Following major surgical procedures, the incidence of pulmonary embolism varies between 0 5 and 8 per cent, the length of the operation, blood loss, hypotension, hemoconcentration, previous thromboembolism and carcinomatosis are some of the factors which contribute to its increased occurrence After ligation of the saphenous vein, it has occurred in a 1 2000 ratio on our service

Two points need emphasis The first, made by Towbin, is that vital statistics, clinical surveys and autopsy material of hospitals do not give the true incidence of this form of terminal illness in the general population.[74] At the Columbus State Hospital, pulmonary embolism was found in 25 7 per

cent of 512 autopsies, with a marked increase in the older age groups This terminal or perhaps agonal state however is not the complication en countered in a hospitalized patient in whom the incidence shows considerable variation, but in a much lower range [75]

The second point is that in spite of the increased attention paid and intensive preventive measures applied to pulmonary embolism its incidence has not been lowered even in institutions especially aware of the problem [76] More than 50 per cent of pulmonary emboli occur without any previous warning and while an endangered group of aged, carcinomatous obese hyperlipemic or fibrillating individuals is well delineated, the acute pulmonary artery occlusion may develop in young, healthy individuals after trauma or after a hernia operation under local anesthesia For this reason intensive *treatment* of embolism needs to be stressed

The Mechanism of Pulmonary Embolism

The literature on this intriguing subject is vast, but mainly two causes have been postulated The *mechanical* theory is that of a sudden pulmonary hypertension with an acute cor pulmonale followed by insufficient return of blood to the left heart and systemic circulation anoxia of the brain, loss of consciousness coronary insufficiency fatal cardiac arrhythmia and death Unquestionably the massive pulmonary embolus which is found in the major branches or main trunk of the pulmonary artery must produce a sudden right ventricular strain In 1939 we demonstrated this in the dog



FIG 188 Flat chest films of a dog before (A) and within 10 seconds after (B) the production of pulmonary embolism. Note the globular appearance and the more transverse position of the heart shadow after the experimental production of embolism. The diaphragm rises. (de Takats, G et al. Pulmonary Embolism Surgery 6 339 1939)



FIG 189 Visualization of the right heart and pulmonary artery before and after the experimental production of pulmonary embolism The right heart and the branches of the pulmonary artery are seen in (a) In (b), after the production of the embolus, there is a dilatation of the right heart, a block of the pulmonary artery and marked venous reflux into superior and inferior venae cavae In (c), 30 seconds later, some blood is going through the main pulmonary artery, but the caval hypertension and reflux is still maintained (Jesser and de Takats Visualization of Pulmonary Artery during its Embolic Obstruction Arch Surg, *42* 1034, 1941)

The heart takes on a more globular appearance and shifts its long axis (fig 188) The embolus was produced by injecting a mixture of barium sulfate, iron perchloride and normal salt solution in equal parts into the femoral vein [77]

There seems to be no argument about this mechanical obstruction being the cause of symptoms and death in pulmonary embolism, except that a number of patients are brought to autopsy on whom the mechanical obstruction is inadequate to explain the sudden shocklike state and death The emboli in such a case may be multiple and peripheral The evidence that there are *autonomic reflexes* at play has been summarized by Griffin and his coworkers, although these authors found no evidence of such reflexes [78]

The acute right ventricular hypertension produces a reflux into the two inflow tracts, namely into the superior and inferior vena cavae (fig 189) Clinically this is recognizable by an increase in the size of the right heart, by venous distention in the neck and by a palpably congested liver The significance of these findings for the proper timing of a pulmonary embolectomy will be discussed on page 307

The effect of epinephrine on pulmonary arterial resistance was also studied by angiocardiograms In figure 190 exposure was made three seconds before (a) and two minutes after (b) the intravenous injection of 1 cc of a 1 1000 solution of epinephrine Note the contraction of the right auricle, the potent reflux of opaque solution into both venae cavae and the enlargement of the pulmonary conus Epinephrine has killed some patients in the acute phase of pulmonary embolism by increasing pulmonary arterial resistance and producing pulmonary edema

The effect of pulmonary embolism on the bronchial tree has been studied in another set of experiments [80] After a control bronchogram, a pulmonary embolus was produced which resulted in marked scattering of the

opaque material as if someone had squeezed the lung by hand Five minutes later the bronchographic pattern is slightly restored In figure 191 there is patchy emphysema in both films after embolism due to incomplete bronchial obstruction, but when the animal was prepared with intravenous atropine and papaverine the bronchographic pattern did not change (fig 192)

We are thus convinced based on our early experimental[79, 80] work on clinical observations since accumulated and on more recent experimental studies, that whenever a partial occlusion of the pulmonary artery or its branches occurs, reflex effects on the heart bronchi on the unobstructed pulmonary arterial bed and even on the upper gastrointestinal tract are demonstrable and contribute to the morbidity and mortality of pulmonary embolism. Since the emergency treatment of pulmonary embolism rests on such an assumption the evidence for such reflex effects are here briefly summarized

In the first place there are well developed baroceptor nerves in the pulmonary artery analogous to those in the carotid sinus which mediate depressor reflexes whenever acute pulmonary hypertension develops A nerve plexus rich in ganglion cells and chromaffin cells exists between the pulmonary artery and the ascending aorta with afferent fibers in the vagi and sympathetics [81] This *glomus pulmonum* is acutely stimulated during total or subtotal obstruction of the pulmonary artery and may contribute to the acute hypotension It was particularly studied by Schwiegk [82] who could produce a sudden drop in blood pressure every time he raised pulmonary arterial pressure This hypotension could be abolished when vagal fibers from the lung were sectioned [82]

While a number of observers could not duplicate these experiments the clinical fact remains that patients may go into a cold bradycardic hypotension followed by death with only a partial obstruction of the pulmonary artery or just a small infarct in the periphery Jesser and I[83] published some

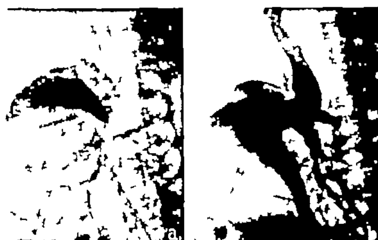


FIG 190 The effect of intravenous epinephrine on pulmonary arterial resistance and vasoconstriction Note the potent reflux into both cavae. This supports the contention that the pulmonary vascular system possesses a potent vasoconstrictor mechanism, and finds a therapeutic application in stellate blocks for pulmonary embolism.



FIG 191 The effect of pulmonary embolism on the bronchial tree of the dog (A) before, (B) immediately after, and (C) five minutes after the production of embolism The opaque material has been squeezed into the radicals and patchy emphysema developed After five minutes there is partial restoration of the pre-embolic bronchial pattern In control experiments, cyanosis and dyspnea did not produce such a response (Jesser and de Takats. Bronchial Factor in Pulmonary Embolism Surgery, *12* 541, 1942)

case reports of fatal pulmonary embolisms in which autopsy showed small peripheral infarcts, therefore a "reflex death" had to be postulated The receptors here are in the visceral pleura or in the lung tissue itself, whose stimulation has wide-spread effects on cardiac rhythm, coronary circulation pulmonary arterial circulation and bronchial tone (fig 193)

There are, of course a number of factors which will accentuate these reflex phenomena Anoxia is one of them and so are certain drugs, such as digitalis morphine cyclopropane and acetylcholine, which have parasympa thetic effects Conversely Nembutal or deep ether anesthesia will inhibit these reflexes and hence it is very rare to see them in pulmonary surgery although they do occur [84] Most physiologists have minimized these reflexes because vagal section does not abolish them However two points may be



FIG 192. The protective effect of 1/75 gr of atropine and ½ gr of papaverine on the bronchial response to pulmonary embolism (A) before (B) five minutes after intravenous atropine and papaverine (C) immediately after and (D) four minutes after the production of an embolus. The patterns are essentially unchanged. (Jesser and de Takats Bronchial Factor in Pulmonary Embolism. Surgery *12* 541 1942.)

made in this connection First, local cocainization of the pulmonary hilus is quite effective, so that the reflexes may be axonic in nature, and second, when the parasympathetic denervation of the lung is complete, so that the Hering-Breuer reflexes are abolished, an average decrease of 46 5 per cent of pulmonary arterial pressure can be obtained [85] And finally, L. N. Katz, whose school has always opposed any emphasis on neurovascular phenomena in pulmonary embolism, has recently published good evidence that a neurogenic or neurohormonal factor is quite active in the production of pul-

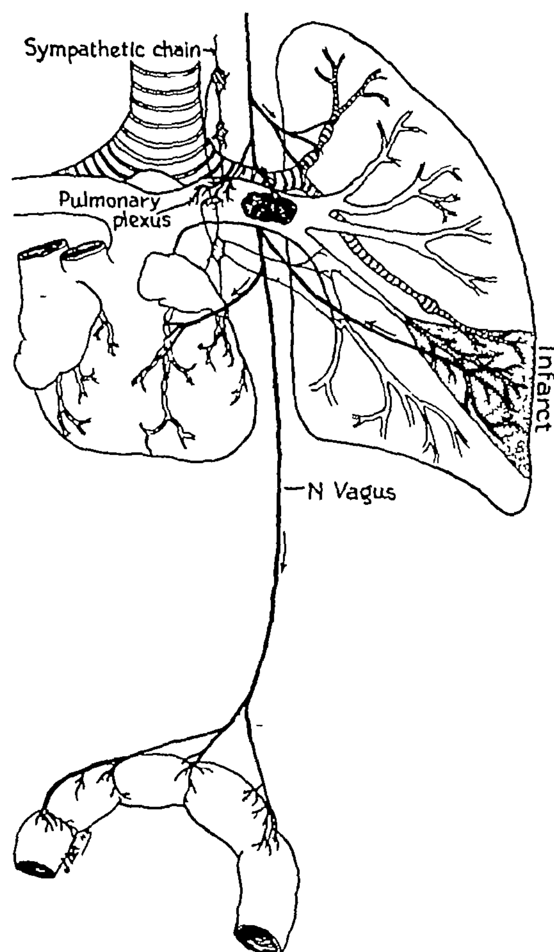


FIG 193 Diagram of reflexes originating from pulmonary arterial obstruction or infarcted lung tissue The afferent impulses travel mainly through the vagus and radiate back as bronchoconstrictor and bronchosecretory fibers They produce vagal inhibition of the heart and decrease coronary flow The dilatation of the right heart produces stimulation of the sympathetic vasoconstrictors (Bainbridge reflex) Spasm of and peristalsis of the upper gastrointestinal tract occur (de Takats, G , et al Pulmonary Embolism Surgery, *6* 339, 1939

monary edema following experimental pulmonary embolism with starch granules [86] I emphasized this mechanism here since the emergency treatment of pulmonary embolism, to be outlined on page 305, is based on this concept

Just what makes the clot break loose is another intriguing problem Mayne, Petersen and I published some data on the meteorologic factors in pulmonary embolism [87] As far back as in the writings of Hippocrates, one finds the idea that the human body is a "cosmic resonator" responding with varying intensity to changes in the meteorologic environment For the vascular tree, this means spasm and relaxation, rise and fall in pressure, and changes in permeability and velocity of blood flow Figure 194 illustrates the trend prevalent in the Chicago area, namely the increase in emboli during the spring and fall and the accumulation of these accidents in the months

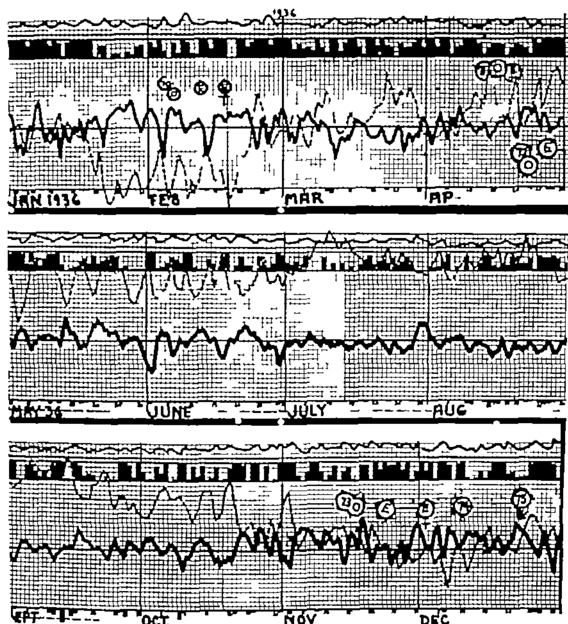


FIG 194 Chicago meteorogram for the year 1936 The upper curve is the wind velocity in miles per hour the black columns are the percentage of sunshine the heavy line is the barometric pressure the dotted line is the mean daily temperature the black columns under the date line are precipitation. The case number is in circles O is operation E is embolism + is death (de Takats, Mayne and Petersen Meteorologic Factor in Pulmonary Embolism Surgery 7 819 1940)

during which the barometric pressure and temperature are markedly fluctuating. Cold waves seemed particularly significant

Pathologic Considerations

Obstruction of the pulmonary artery or its branches may result in various findings in the lung parenchyma which they feed. The obstruction may be due to embolism but sometimes to thrombosis in situ recently much interest has been shown in massive thrombotic occlusion of the large pulmonary arteries [87b] Even on the autopsy table the differentiation between em

bolism and thrombosis is difficult, since a small undetected embolus may initiate a large thrombus In Table VI the criteria of differentiating primary from secondary thrombosis are listed.

Table VI

CRITERIA FOR THE DIFFERENTIATION OF PRIMARY AND SECONDARY THROMBOSIS IN THE PULMONARY ARTERY*

PRIMARY THROMBOSIS	THROMBOSIS SECONDARY TO EMBOLISM
Local disease of pulmonary artery.	No local disease in the pulmonary artery
Thrombus is a cast of the vessel	The embolus is usually coiled
Often there is lamination of the clot in layers, parallel to the arterial wall	The source of the embolus is often found and corresponds to the shape of the clot in the pulmonary artery.

* From Ball, Goodwin and Harrison Massive Thrombotic Occlusion of the Large Pulmonary Arteries Circulation, *14* 766, 1956

Since over one half of the patients manifesting the symptoms of pulmonary embolism show no warning in the periphery, and since even at autopsy at least 10 per cent of the cases show no source of the pulmonary embolus, one may suspect a certain small number of cases to originate in the pulmonary artery, particularly in the presence of mitral stenosis or pulmonary hypertension of other origin

Pulmonary embolism is, of course, not equivalent to pulmonary infarction There may be no infarction at all when the bronchial arterial system serves as a collateral and pulmonary venous drainage is adequate, there may be incomplete infarction with localized venous congestion and edema, there is complete infarction especially when pulmonary venous pressure is high and the bronchial arterial circulation cannot reach the infarcted parenchyma [88] When infection, decreased aeration due to atelectasis or congestion is present in the lung, pulmonary infarction is more likely to occur [89] This needs emphasis, since many residents still have to see hemoptysis and a shadow in the lung, preferably triangular, before they diagnose pulmonary embolism In the absence of hemorrhagic infarcts, there may be anemic ones which are translucent in the roentgenogram, especially if bronchostenosis is present Then, again, one can find an infarct without any evidence of pulmonary embolism, the clot having been absorbed or at least partially recanalized

Repeated and partially recanalized emboli and their effect on the parenchyma of the lung have been carefully studied by Belt [89] He showed that the primary occluding embolus may grow proximally and distally and this is a good reason for early intensive anticoagulant therapy Restoration of the lumen does occur and can be observed experimentally,[90] but it undoubtedly leaves an arterial stenosis with some functional damage

The hemorrhagic infarct gradually organizes shrinks becomes aerated and may leave only some linear opacities visible on roentgenogram If the clot is infected a septic infarct may develop with lung abscess or gangrene in its wake With intensive antibiotic therapy these complications are mostly though not always, preventable

With the infarct extending to the visceral pleura pleural effusion which is occasionally hemorrhagic is the rule the exudate is fibrinous and is re sponsible for the friction rub most often audible at the right base

Clinical Symptoms and Signs

Pulmonary embolism occurs in at least four recognizable forms and obviously transitions from one form to another are frequent

(a) THE SUSPECT OR SUBCLINICAL EMBOLUS Such a patient either ambulatory or bedridden in a post traumatic postoperative or postpartum state after a coronary thrombosis or auricular fibrillation develops a short period of dyspnea, perspiration and tachycardia. There are no physical find ings chest pain fever roentgenologic or cardiographic changes are absent There are no localized thrombi in the peripheral veins

The symptoms may never recur but the significance of this form lies in the fact that it may be the initial small shower to be followed by a larger embolus or in a few days small streaks of rusty sputum or a mild friction rub occur in which case a clear picture of the second form is present.

(b) THE SMALL PULMONARY INFARCT Many of these are ignored or misdiagnosed They may be single or multiple and if they reach the visceral pleura, dry pleurisy pleurisy with effusion or atypical (virus) pneumonia is diagnosed More than half of them appear without any obvious thrombus in the peripheral veins The right lower lobe of the lung is most frequently *affected, followed by the left lower lobe the left upper lobe and, least fre-*quently the right upper lobe Infarcts of the lower lobes are much more pain ful and present the picture of diaphragmatic pleurisy with splinting of the diaphragm, pain on breathing and shoulder pain due to irritation of the phrenic nerve

On physical examination one finds absent or diminished breath sounds dullness on percussion pleuritic rub or intrapleural fluid Atelectasis or atelectatic pneumonia may supervene The atelectasis is partly of reflex nature the concept of reflex atelectasis, which has also been utilized to explain the sequelae of trauma to the chest has been very fruitful [91] The bronchial tree is capable of contraction and pours out a massive secretion of mucus immediately following pulmonary embolism. This also happens after rib fracture and helps to explain the wet lung" described during the Second World War [92] Certainly the postoperative bronchopneumonia, so often feared in the past is partly embolic in origin.

(c) THE MASSIVE SUBLETHAL, PULMONARY EMBOLUS The patient is suddenly stricken with an obviously serious vascular accident He is cold clammy pale or cyanotic, and dyspnea is not as prominent as syncope The

pulse is rapid, feeble and barely palpable The blood pressure is low or may not be obtained

The resemblance of such a condition to a coronary occlusion is striking and a differential diagnosis may be difficult The roentgenologic and cardiographic findings will be given on page 302, but they may not allow a clearcut distinction between the two lesions The acute hypotension, particularly in cerebral arteriosclerotic patients, may produce the symptoms of a cerebral insufficiency and thus give rise to symptoms of a cerebral vascular accident The reflex effects on the gastrointestinal tract simulate gallstone colic and pylorospasm with rectus rigidity, just as coronary occlusion and pulmonary embolism may lead to hasty, ill conceived laparotomies for pseudoabdominal lesions It has been surprising to see how patients tolerate an unnecessary laparotomy under such conditions A chest lesion should always be excluded before such an abdominal exploration is done

Table VII

INITIAL SYMPTOMS AND SIGNS OF PULMONARY EMBOLISM AS NOTED IN THE NURSES' RECORDS IN 100 CASES*

Symptom	Cases	Symptom	Cases
Dyspnea	42	Weak Rapid Pulse	26
Chest pain	32	Shock	12
Cyanosis	24	Restlessness	9
Nausea and Vomiting	6		
Pain in Abdomen	5		
Chill	4		
Convulsion	3		
Dizziness	2		

Four patients were found dead

The size of the infarct, which presents itself in the next few days after the critical period is over, is by no means commensurate with the alarming symptoms Large infarcts may give rise to severe pain, dyspnea and cyanosis but without the acute collapse On the other hand, multiple small peripheral emboli, reaching suddenly and additively the reflexogenic zones of the visceral pleura, may produce the syncopal type of symptoms with pallor and acute hypotension To us, this represents a reflex effect and should be so treated

(c) THE FATAL PULMONARY EMBOLUS In our first clinical report with Jesser,[83] it was found that 8 5 per cent of 70 cases of fatal pulmonary embolism died in less than 10 minutes This figure obviously varies depending on the type of material My former associate, Matthew H Evoy, collected 1,000 cases from the literature[93] and found a much higher percentage of death within 10 minutes, namely 46 per cent As he himself pointed out, the percentage of survivals may be higher than this since the time of onset may go unnoticed, depending on the nursing care The notation on the chart, "died

* From de Takats G and Jesser, J H Pulmonary Embolism, Suggestions for its Prognosis, Prevention and Management J A M A, *114* 1415, 1940

suddenly many times actually means found dead suddenly and the excellent nurses records at old St Luke s Hospital* have been extensively utilized in recording the initial symptoms and signs of pulmonary embolism (Table VII)

The early symptoms and signs need emphasis since there may be more time than many people suppose to institute emergency measures which seem to have transformed a patient with severe vascular collapse into one *with a painful infarct in one lobe*



FIG 195 Six days after lumbar sympathectomy for Buerger's disease a sudden right sided pain developed at the costal margin radiating to the shoulder The film shows an elevation of the right leaf of the diaphragm with haziness in the costophrenic sinus. Note retraction of the right half of the chest the ribs are closer to each other No cough or hemoptysis developed.

The patient then either dies suddenly or recovers There is however the important syndrome of the slowly fatal pulmonary embolus described by Robin Pilcher [94] After surviving the initial impact, these patients die of a progressive dilatation and failure of the right heart Pilcher suggested that patients showing no improvement, but a progressive increase in cor pulmonale with distention of neck veins should be operated on within two to three hours after the onset of symptoms 60 per cent of our studied patients lived longer than one hour and many patients (24 out of 70) died in one to

* About to merge with Presbyterian Hospital at this writing.

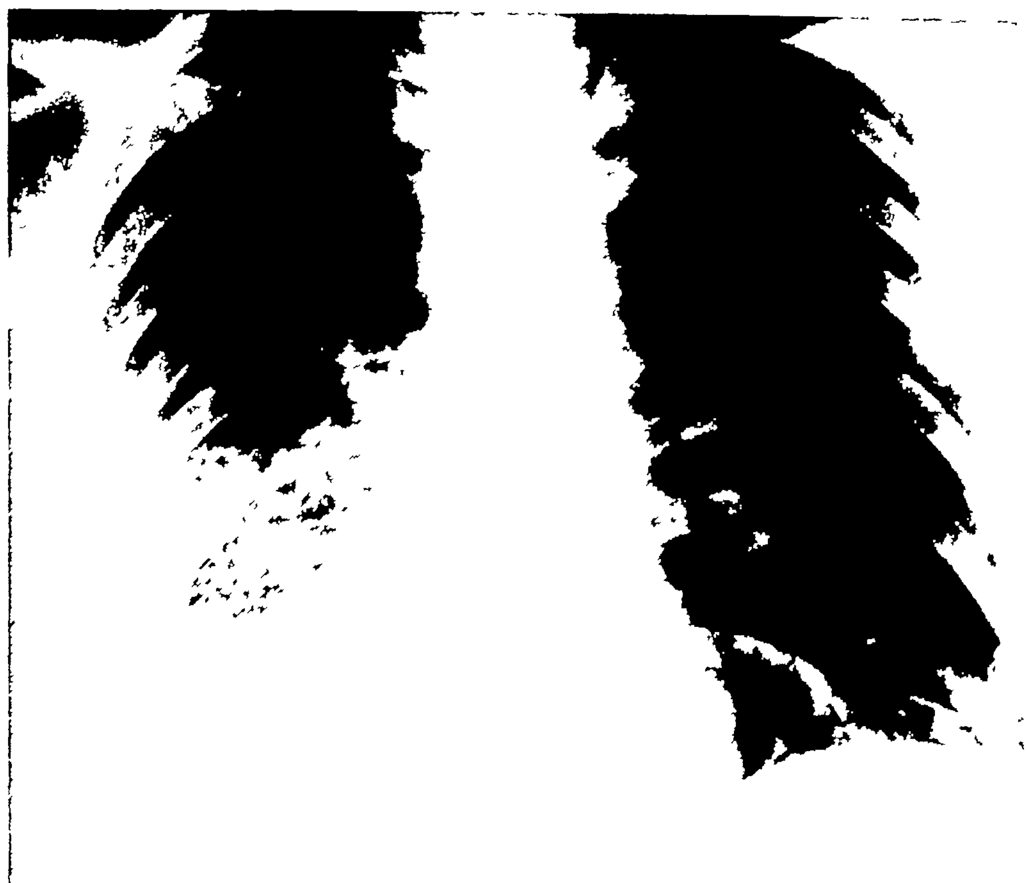


FIG 196 Fluid level, patchy atelectasis and consolidation in the left lower lobe six days after hysterectomy In the original film, areas of translucence were visible as a result of bronchostenosis (Westermark's sign)

several days The extremely limited indications for embolectomy will be discussed under treatment (p 307)

Radiologic Findings

The earliest roentgenologic findings in pulmonary embolism are an elevation and splinting of the diaphragm on the affected side (fig 195) It takes three to four days before a patchy atelectasis, a pleural effusion or a hazy consolidation shows in a portable roentgen film (fig 196) The wedge-shaped infarct is the great exception [95] Occasionally, multiple infarcts may look like malignant metastases and such a case was seen after prostatic resection for carcinoma of the prostate The patient died of pulmonary embolism and there were no metastases to the lung at autopsy In at least three instances, I have seen massive pulmonary infarcts resected for bronchogenic carcinoma of the lung, because of a roentgenologic appearance which was highly misleading

The point should be made that the roentgen film is of great help in the diagnosis and management of pulmonary embolism, but naturally its interpretation must be correlated with clinical observations Repeated pleural effusions must be suspected of having an embolic background and many experienced internists have missed the diagnosis

The Electrocardiogram

The concept that the acute cor pulmonale produces a characteristic right ventricular strain pattern and is distinct from the pattern of coronary occlusion has been brought forward by noted cardiologists [96] This consists of an electrocardiographic pattern of insufficient right coronary flow resulting in ischemia of the right ventricle and the posterior surface of the left ventricle there is an abnormally deep S and Q_3 wave Nothing in our studies with George K Fenn[77] would indicate, however that either experimental or clinical pulmonary embolism has a characteristic cardiographic pattern since the reflex effects on the myocardium and the general hypoxia resulting from the acute hypotension result in patterns of acute coronary occlusion In figure 197 (p 305) serial tracings are shown of a 33 year old woman who developed pulmonary embolism 10 days after cholecystectomy The idea that the abnormal pattern reverts to normal as soon as the acute cor pulmonale disappears is wrong, and there are definite infarcted areas in the heart as a result of a temporary coronary insufficiency Such histories reinforced by autopsy evidence have been reported from our clinic [83]

For this reason the cardiogram alone can seldom be used to differentiate between pulmonary embolism and coronary occlusion in serial determinations one may detect a deterioration of the electrocardiogram as extension of the pulmonary thrombus or a recurrent embolus takes place This may happen with little clinical evidence except perhaps a slight rise in pulse or temperature

The Clotting Mechanism

If one follows the course of pulmonary embolism with serial determinations of clotting time or prothrombin activity a type of three phasic curve is observed, such as one sees after operations Since most patients are on anticoagulants the untreated cases are rare but occasionally it has been possible to obtain a postembolic curve in the absence of anticoagulant therapy Surprisingly enough there is a spontaneous hypocoagulability shortly after a massive embolus Liver damage fibrinolysis and mobilization of heparinoid

Table VIII

SPONTANEOUS HYPOPROTHROMBINEMIA AFTER PULMONARY EMBOLISM

DATE	PER CENT OF PROTHROMBIN ACTIVITY	REMARKS
June 20 1946	85	Convalescing from coronary occlusion.
June 21 1946	60	Pulmonary infarct superficial femoral vein ligated under local anesthesia.
June 22, 1946	46	No anticoagulants were given.
June 23 1946	50	
June 24 1946	70	
June 26, 1946	66	

substances or of antithrombin may be at play singly or in combination. Such a prothrombin curve has been seen in an elderly physician who was recovering from a coronary occlusion and developed a pulmonary infarct (Table VIII).[97]

The stress response of the clotting mechanism has already been discussed in a previous chapter (pp 31-33), in addition, a massive thrombus may use up a lot of fibrinogen and thus defibrinate the blood. If anticoagulants are given at such a stage, bleeding may result Thus, serial determinations of clotting time or prothrombin activity should always be available if anticoagulant therapy is used

Treatment of Pulmonary Embolism

No matter whether the pulmonary embolism is suspected or whether there is a small or massive infarct, intensive heparin therapy is the basis of all treatment As emphasized on page 282, insufficient treatment is useless and may even protract the disease Heparin therapy should always be started by intravenous injections, giving at least 100 mg four to six times a day Clotting times need to be done once a day, preferably before the administration of the morning dose of heparin to avoid a step ladder type of rise of clotting time following each injection This is a safer method than the continuous drip, because the latter needs continuous supervision and readjustment of the rate of flow Besides, a sudden massive dose of heparin has a demonstrably better effect on an acute soft thrombus than a slow continuous drip

The intermittent intravenous heparin therapy should be kept up until defervescence, slowing of the pulse and diuresis indicate that the acute thromboembolic episode has reached the second stage of the adaptation syndrome, namely that of resistance [7] At this time, one can begin subcutaneous injections of 10 per cent or 20 per cent heparin in aqueous solution, two to three 100 mg doses suffice to keep the one-tube Lee-White clotting time at 10 to 15 minutes It is important to maintain anticoagulant therapy for *at least three weeks*, if this rule is broken, as we have done from time to time, a recurrent embolus or a propagation of the first embolus occurs in an alarming number of cases Equally objectionable is the use of subcutaneous heparin therapy without the preliminary saturation of the patient with large intravenous doses When such a method is used, thromboembolic phenomena occur or may even be facilitated during heparin therapy

The purposes of intensive heparin therapy are. (1) to prevent both the primary thrombus and the pulmonary embolus from propagating, (2) to facilitate the canalization of the embolus which occurs spontaneously, and (3) to aid the fibrinolysis in the infarcted lung [98] The danger of increasing the hemorrhage in the infarct is slight, if existent at all. Actually, it is better for the patient to cough up unclotted blood from the bronchi than to have it accumulate in the form of mucous and clotted plugs in the bronchi causing atelectasis.

There are emergency measures however which should *precede* this basic treatment if the patient is in acute hypotension or exhibits severe dyspnea Based on the previously outlined animal experiments, oxygen by mask or catheter 1/75 to 1/60 gr of atropine given intravenously and ½ gr of papaverine given intravenously should be administered *early and without fail*

The following history is illustrative [83]

Mrs. E.T., a 33 year old woman, developed a sudden hypotension 10 days after cholecystectomy Her blood pressure fell from 120/80 mm. to 70/58 mm. of mercury 1/75 gr of atropine and ½ gr of papaverine were given within 30 minutes after the onset of symptoms. The patient s pulse immediately became stronger and she emerged from a seemingly moribund state.

Oxygen was started which relieved her dyspnea. 17 days later after an afebrile period of seven days, a second pulmonary embolism occurred which was again aborted with atropine and papaverine Roentgen films confirmed the diagnosis of pulmonary embolism Electrocardiograms revealed the evolution of a coronary insufficiency (fig. 197)

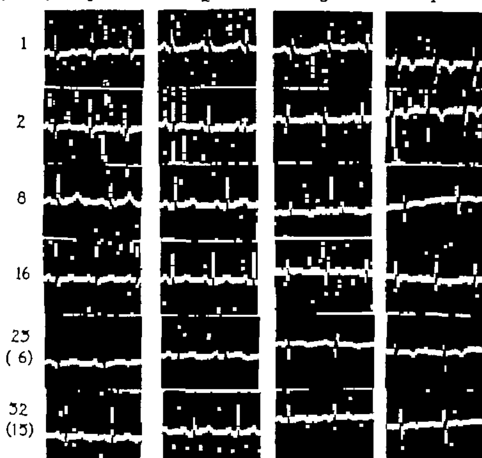


FIG 197 Serial electrocardiograms in Mrs. E.T., 33 years old, who developed pulmonary infarcts 10 days and 17 days after cholecystectomy Note that after the initial improvement, T waves became more depressed again 23 days after the first embolus. This corresponded to the second infarct She had full functional recovery with minimal leg signs.

mation or a stenosing atheromatous lesion creates an area of increased vulnerability of the arterial wall. The segmental arterial thrombosis in thromboangiitis obliterans, in trauma and as a terminal lesion in stenosing atheromatous lesions is frequent. A fall in blood pressure as a result of trauma, hemorrhage, anesthesia or coronary occlusion is often the precipitating factor in the acute closure of the vessel; in fact, since roughly a 70 per cent constriction of the arterial lumen is necessary to produce clinical symptoms, often the acute thrombosis is the first indication of vascular disease.

Ligation of an artery may or may not cause an ascending or descending thrombosis, depending on the available collaterals and thus on the prevailing pressures in the proximal and distal segments. Wherever possible, an artery should be ligated just distal to a collateral branch. With the widespread use of arterial sutures, ligation of large arteries is practiced less frequently.

Aneurysmal sacks invariably contain mural thrombi, but these may become totally occlusive, obstructing the lower end of the sack. This is especially true in popliteal aneurysms. Aortic aneurysms, on the other hand, may liberate thrombi—and also plaques—distally and cause acute ischemia.

Whether or not there is such a thing as a simple arterial thrombosis, meaning that a healthy segment of an artery can close without previous trauma, disease or embolic phenomenon, is open to question. Whenever the pathologist reports a normal vascular wall with little if any attempt at organization, one naturally wonders about a lesion above or below the sectioned arterial segment. The constricting effect of tendons and ligaments in Hunter's canal, at the upper end of the soleus muscle or at the thoracic outlet have been commented on elsewhere (p. 26). Platelet deposits in areas of turbulence, as first emphasized by Aschoff,[34] are instrumental in initiating thrombosis.

Arterial Embolism

On the other hand, arterial embolism results from the liberation of a thrombus from the heart or from the wall of a thrombotic artery. The embolus usually lodges at bifurcations, and those lodged at the aortic, common femoral and popliteal bifurcations are dangerous. The vessel plugged by the embolus hugs it by muscular contraction which may later be released, thus allowing a peripheral migration of the embolus. This may result in the reappearance of a previously absent pulse, but a more terminal occlusion of digital or muscle vessels may not necessarily result in an improvement in circulation.

The occluding clot, whether due to thrombus or embolus, shows varying grades of recanalization. A clot may become organized by being invaded by the vasa vasorum and turned into a fibrous cord. This sets up an adventitial reaction and actually, as Leriche has postulated, becomes the origin of vasomotor reflexes. Leriche based his arterectomies on the premise that an area of nerves and nerve endings caught in the adventitial reaction are responsible for the vasomotor phenomena in a limb suffering from chronic arterial occlusion.[106] As the organization proceeds, the vessel gets smaller

and shows longitudinal contraction which according to Palma,[107] adds to collateral embarrassment and is relieved by transecting the artery and letting the two ends retract While this is a novel and interesting thought it is doubtful whether release of the tension can ever deliver as much blood as an endarterectomy or a bypass Certainly a simple transection of a permanently occluded artery can do no harm and probably little good

Of far more interest is the fact that thrombosed arteries recanalize more often in thromboangiitis than in atheromatous occlusions and frequently than in embolism This lacunar canalization (Dible[18]) may just be a microscopic finding, or may actually carry blood through a merger of many small endothelially lined channels If one knew the exact mechanism of such recanalizations the natural processes might be aided by surgical and other means

The mechanical pounding of pulsating blood above a fresh occluding thrombus might be effective but against this theory speaks the fact that pulmonary emboli with much lower pressures frequently recanalize Clot retraction takes place not only in the test tube but in a vessel recently occluded by a thrombus Buditz-Olsen[108] has made a thorough investigation of clot retraction it is a special function of platelets is inversely proportional to the fibrinogen content of the plasma and is influenced by the volume of cells expressed by the hematocrit Buditz Olsen concluded that the force of clot retraction is so small that it becomes extremely unlikely that it serves any physiological purpose This leads us to fibrinolysis as being the potent source of recanalization

I have already alluded to the importance of fibrinolytic activity in the spontaneous dissolution of clots and efforts to bring activated fibrinolysin (plasmin) in close contact with fresh clots have been cited in chapter 4 Blood clotting. From a practical standpoint, it is obvious that in order to effect lysis of the clot it must be fresh it must be in close contact with high concentrations of the enzyme and the enzyme must not digest anything else but the thrombus

Clinical Symptoms of Arterial Thromboembolism

The symptoms are those of an acute arterial occlusion The limb is pale and cold the veins are empty and the arteries do not pulsate There are motor and sensory disturbances The ischemic pain is severe aggravated by heat and elevation and relieved by a cold pack or by dependency In arterial emboli of the younger rheumatic age group whose fibrillating auricle contains thrombi and is the source of embolism, diffuse vasospasm may mislead the examiner in regard to the site of occlusion Thus it is wise to spend an hour or so in trying to relieve the vessel spasm, since otherwise a femoral exploration may be done in case of an embolus to the anterior tibial artery Many patients with rapid auricular fibrillation and after excessive mercurial diuresis, throw minor emboli which leave them with lost pulses but little if any functional disability It is important to be aware of such minor emboli

preceding the major one, to be treated surgically, since the pulses may never return even after the fresh embolus has been totally extracted.

The symptoms of an acute arterial thrombosis, superimposed on a previously recognized or unrecognized arterial disease, are identical with those of an embolus. Differentiation between these two types of arterial occlusion is sometimes impossible. Obviously if the patient has known heart disease, if his heart fibrillates and if his episode is quite sudden with no previous symptoms of vascular disease in the extremities, then the diagnosis of embolism is clear. The fact, however, that the patient has had a coronary thrombosis followed within a few days by ischemia of an extremity by no means indicates that a mural thrombus has been detached from the left ventricle: he could have been in shock, and during the acute hypotension a stenosing atheroma of a major artery became an occlusive one, with a small thrombus closing the narrowed segment. The treatment is always directed against the acute arterial obstruction, and the old argument as to whether the obstruction is due to thrombus or embolus is of less importance than whether or not restoration of circulation is possible.

The Levels of Occlusion

It is customary to state that emboli get stuck at bifurcations and thrombi, if based on atheromata, are more often at points of mechanical stress, such as in Hunter's canal, in the common iliacs or at popliteal level. This is purely an academic distinction, because the iliac stenosis, when it becomes obstructive, will immediately produce ascending or descending thrombi, thus clouding the picture.

With both femoral arterial pulsations absent, with a palpable or audible bruit at the level of the navel and with pale and paralyzed extremities which are painful and yet hypesthetic, the diagnosis of an occlusion at the aortic bifurcation is clear. When one femoral artery pulsates normally and the other is absent, an iliac arterial occlusion is present. Sometimes one feels the powerful longitudinal thrust of a short iliofemoral embolus and mistakes this for a patent femoral artery, only to find that it is the clot that pulsates. This means, however, that one will have little trouble in extracting the proximal part of the embolus.

When both femoral arteries are freely pulsatile but one extremity is cold, pulseless and paralyzed, the skin temperatures may help to decide the level of occlusion. Because of available collateral circulation and after vessel spasm has been relieved, a sudden drop in temperature is always a handwidth or more below the actual site of occlusion (fig. 198). Thus, a common femoral arterial block will produce a temperature drop at midthigh, and a popliteal embolus will cool the limb to the upper third of the lower leg. One may localize the embolus with an oscillometer or even with an arteriogram. However, the routine use of arteriograms for the sake of localizing the level of obstruction is unattractive, since it may well hasten the propagation of a thrombus.

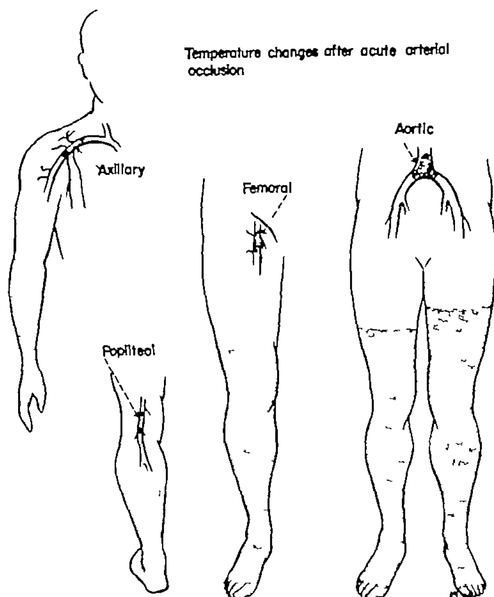


FIG 198 Diagram of temperature levels following different sites of acute arterial occlusion. Note that the shaded area (drop in skin temperature) is well below the level of occlusion.

Nevertheless an occasional carefully timed arteriogram, done with never higher than 35 per cent Diodrast and preceded by 10 cc of 1 per cent procaine mixed with 10 mg. of heparin will yield instructive pictures In figure 199 a tourniquet left on for one hour in a middle-aged sclerotic patient resulted in a segmental thrombosis with little evidence of damage above or below the constriction this is an ideal case for restoration of continuity

In another patient the acute popliteal thrombosis (fig. 200), which was treated by sympathectomy ascended to midthigh six weeks later necessitating amputation

The Propagation of Arterial Thrombi or Emboli

In a 43 year old woman seen only a week after she suffered a right iliofemoral embolus from a rheumatic heart with mitral stenosis and auricular fibrillation, a soft thrombus could be extracted under local anesthesia

preceding the major one, to be treated surgically, since the pulses may never return even after the fresh embolus has been totally extracted

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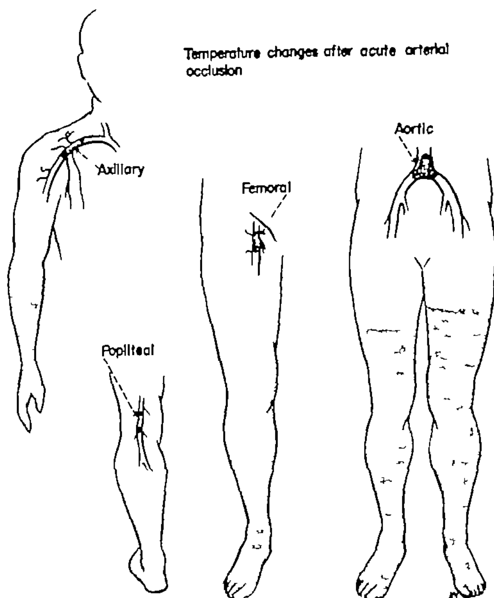


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FIG 199



FIG 200

FIG 199 Arteriogram in a middle-aged patient seen several months after a bunion operation done under a tourniquet She developed a temporary foot-drop and marked claudication The arterial occlusion seems to be limited to the width of the tourniquet There is minimal arteriosclerosis above and below the occlusion, as seen roentgenologically

FIG 200 An acute popliteal thrombosis in an active middle-aged hospital employee who walked many miles daily collecting delinquent accounts The occlusion was sudden and he lost his leg six weeks later when the occlusion reached to midthigh

through an inguinal incision (fig 201) Three months later a successful mitral commissurotomy was done

The initial clot, whether thrombus or embolus, need not be a long one, but it sets up a chain reaction of further apposition of thrombi in both a central and a peripheral direction It is not possible to predict the rate of secondary thrombosis because in some patients this proceeds with great rapidity and causes gangrene, whereas in others, whose blood pressure is well maintained and in whom the normal anticoagulant mechanisms (antithrombins and fibrinolysins) are active, the clot remains localized and may even recanalize

This is the reason why all arterial thromboembolic phenomena require intensive heparin therapy which, however, must be timed to fit in and not interfere with other therapeutic measures

The Management of Arterial Thrombi and Emboli

In the presence of an *acute* arterial occlusion, the following steps are taken to insure a viable and functionally adequate limb·

(1) *Immediate hospitalization is imperative* There is so much to be done under such close supervision that treatment in the home is to be discouraged Transportation by ambulance will hurt the patient much less than leaving him at home Experience has shown again and again that patients who are already in the hospital when the occlusion occurs have a much better chance for successful treatment In metropolitan areas it may take from 6 to 12 hours for the patient to call the doctor for the doctor to come to the patient s house for the patient to decide on hospital care and for the ambulance to get the patient to the hospital From then on the proper timing and sequence of the patient s care is the responsibility of the resident and attending staff

(2) *The cause of the acute arterial occlusion* may be obvious as in the presence of a rapidly fibrillating rheumatic heart after an obvious or silent coronary thrombosis or in case of a palpable popliteal aneurysm which suddenly fills up with a clot But the cause may remain obscure for a while or forever and one should not subject the patient to a lot of unnecessary procedures to determine the cause of the occlusion A cardiogram should be taken if there is time for Lary and I have reported on a series of ambulatory silent myocardial infarctions in whom the first symptom was a peripheral arterial embolus [109]



FIG 201 Margaret B., aged 43 with known mitral stenosis and auricular fibrillation, threw an embolus to the right femoral bifurcation. In a week the clot extended almost to the aortic bifurcation. Late embolectomy successfully freed this segment, and mitral commissurotomy by Dr O C Julian eliminated the source of the embolus.

In addition to 27 patients with peripheral arterial emboli whose charts we reviewed (Table IX), we recognized eight patients who were ambulatory, despite recent coronary occlusions previously unrecognized One of them had a cholecystectomy done elsewhere for the upper abdominal pain of the myocardial infarct

The only purpose of a cardiogram in the presence of an acute arterial occlusion is to rule out or discover a fresh myocardial infarct The more recent determinations of transaminase are of no help here, since a high level

Table IX

SOURCE OF ARTERIAL EMBOLI OF THE EXTREMITIES*

SOURCE	NO OF PATIENTS	AVERAGE AGE IN YEARS
Rheumatic heart disease	6	45
Myocardial infarction	7	53
Bacterial endocarditis	3	39
Arteriosclerotic heart disease	2	68
Aneurysmal clot	2	64
Atheromatous plaque	3	74
Unknown	4	50
Total	27	

* (Lary, B G and de Takats, G Peripheral Arterial Embolism after Myocardial Infarction J A M A , *155* 10, 1954)

of this enzyme can be obtained in acute muscle ischemia of the leg in the absence of myocardial infarction The knowledge of the presence of a recent myocardial infarct will influence the selection of anesthesia or the magnitude of surgical procedure

A transabdominal extraction of a saddle embolus of the aorta in the presence of a 10 day old myocardial infarct, or during congestive heart failure, may have to be modified to a simpler, retrograde extraction through both femoral arteries under local anesthesia

More and more acute thromboses superimposed on stenosing atheromatous plaques are being recognized They too need exploration, and the dictum of "no surgical extraction of arterial thrombi" definitely requires modification

(3) *The extent of vasospasm* needs to be determined and may mask the site of the organic occlusion The surgeon may not be as often confronted with this problem as the physician who first sees the patient, or the resident Again and again one may see an embolus to the posterior tibial artery produce a pulseless femoral artery in the groin, with a cold, cyanotic extremity

to the middle third of the thigh Deep venous thrombosis a fracture or a bullet wound grazing or passing close to a major artery can evoke in certain individuals such a massive vasospasm that the site of the organic occlusion is in doubt

For this reason the first step in the treatment of acute arterial occlusions is the release of vasospasm In the earlier years of vascular surgery this spasm was regarded as being entirely neurogenic mediated by the sympathetic nervous system [106] One can unquestionably obtain by sympathetic paralysis a considerable improvement in the color and temperature of an acutely ischemic limb There is also a release of venospasm which results in a de crease of edema if this is present For many years our service has routinely performed paravertebral blocks in all cases of acute arterial ischemia and if there is trained personnel on the vascular surgical or on the anesthesia serv ices to do this simple procedure and to do it *immediately* and *correctly* it should be the first step in the management of arterial occlusions

An abdominal heat cradle to provide reflex vasodilation to the lower extremity has also been our practice in earlier years Donald Miller and I[110] showed that in different grades of arteriosclerosis approximately as much increase of blood flow can be obtained by applying indirect heat through a large abdominal heat cradle as by placing the heat cradle directly to the affected extremity Because direct application of heat even a moderate amount may lead to an increase in pain and acceleration of gangrene *heat should never be applied directly to an ischemic limb* Indirect heat however produces a reflex vasodilation and will not increase the metabolism of the hypoxic cells

Edwin Lehman suggested the use of a large abdominal heat cradle in acute vascular occlusions to overcome the spasm of collateral vessels [111] It should be emphasized however that this reflex vasodilation cannot operate in a sympathectomized extremity or in one whose sympathetic nerve supply has been temporarily blocked with procaine Therefore we have for many years wrapped up the ischemic extremities from toes to groin in rolls of absorbent cotton to prevent dissipation of heat by evaporation and to pre vent pressure sores The ischemic extremity is thus maintained at body temperature if there is any amount of arterial blood reaching it If the limb remains ice cold under the cotton wrap frank gangrene is imminent

It needs however emphasis that muscle spasm in the occluded artery may be myogenic the vessel hugging the clot like a spastic bowel hugs a gallstone

In 1936 the use of intravenous papaverine in acute arterial occlusion was advocated and I reported striking benefit in some cases [112] One half gr doses were given intravenously two to three times a day However should a pulsating artery femoral or subclavian be available above the obstruction intraarterial papaverine in the same dose is preferable

Kinmonth however has since brought forth important evidence to show that the *topical application* of papaverine in 1 to 2 per cent solution will produce relaxation of a spastic artery [113] hot compresses or infiltration of

procaine may not do so While topical papaverine was originally advocated for traumatic spasm, one can make extensive use of this method during vessel anastomoses Autogenous vein grafts, especially, will contract severely on handling, they too relax under papaverine

Holden has also emphasized myogenic spasm in his excellent monograph on acute arterial occlusions [114] He noted marked spasticity of the artery distal to a femoral embolus, which existed under spinal anesthesia and after periarterial stripping was done proximally to the spastic area. Such spasm may persist for several days and undoubtedly contributes to the extent of distal thrombosis As pointed out on page 3, this active contraction of the empty vessel may be an example of Burton's concept of critical closing pressure

(4) *The use of anticoagulants* in acute arterial occlusion is urgent and should promptly follow any procedure to release vasospasm Intra-arterial heparin given into a pulsating arterial segment above the block is the ideal method 10 cc of 1 per cent heparin may be injected into the subclavian or femoral arteries several times in 24 hours Unfortunately, most emboli or thromboses obstruct the very segments which one would wish to inject, so that intravenous administration has to be applied in the same doses and repeated three times a day

In the early days of anticoagulant therapy it was thought that prolonged clotting times should be brought back to normal before any surgical procedure was begun. Therefore, heparin was used sparingly when operative interference was contemplated We have become more confident, however, in operating on patients under anticoagulant therapy, and this principle is especially applicable to embolectomies and thrombectomies 100 mg of heparin given preoperatively or 50 mg given during the operation are safer than giving heparin postoperatively, since it is then that large hematomas are produced Heparin thus should not be withheld, even if surgical exploration is immediately contemplated Dicumarol or any other prothrombin depressant has no place in such acute occlusions Even if given intravenously, as Warfarin, these drugs have a delayed and prolonged action and are out of place here

Whether or not an active fibrinolytic enzyme can be administered intra-arterially to dissolve the fresh clot, as Cliffton has done experimentally,[68] has not yet had sufficient clinical trial Our present use of activated aged plasma containing active plasmin (fibrinolysin) has been outlined on page 288

(5) The localization and surgical removal of the occluding clot (*embolectomy or thrombectomy*) is obviously the most direct and most efficient way to clear an obstructed arterial pathway *The timing of the operation* is most important, however

Obviously, when the diagnosis of an acute arterial occlusion is made, rapid systematic action is imperative Vasospasm is relieved with paravertebral block, with heat cradle and with hot drinks These measures may convert the picture of a high arterial occlusion into a warm, pink, freely moving extremity with normal sensation and return of pulsations, except in a ter-

minal artery of the foot Therefore it is wise to spend an hour or so immediately after the vascular accident in watching the limb and using various methods of vasodilation If the femoral artery pulsates one can inject ½ gr of papaverine (0 03 Gm) 025 Gm of Priscoline or heparin

When there is no restoration of major pulses and the patient is not in shock or frank cardiac failure exploration of the closed artery is indicated While the optimal results in our own material and in that of others are obtained in the first 6 to 24 hours [115] later extraction of clots may be indicated, even as long as a week unless the limb is frankly lost [116]

In my experience the late removal of thrombi as done in a 62 year old patient eight days after a sudden arterial occlusion (fig 202) is possible when the thickened hyalinized intima proceeds very slowly with organization of the clot and when an atheromatous sequestrum can be simultaneously shelled out We have come a long way from trying to make a strict distinction between embolus and thrombus, attempting to remove the former and leaving the latter alone First such a distinction is often impossible Second,



FIG 202. Arteriogram in a 62 year old patient with atheromatous stenosis in Hunter's canal, who developed a sudden arterial occlusion while vacationing in Mexico A long, soft thrombus together with an atheromatous sequestrum was extracted eight days later with restoration of the lower femoral segment. Note a stenosing atheroma above the complete occlusion.

ith the advent of endarterectomy we know that the thrombus can be re-
oved together with the atheromatous inner lining of the vessel, whereas in
e old days the removal of the thrombus was promptly followed by reforma-
on of the clot. And third, in suitable cases and with adequate distal back-
ow, one can resect or bypass such a segment and thus restore continuity
nquestionably, however, embolectomies are more successful than throm-
ectomies

For these reasons we have dealt here with embolectomy and throm-
ectomy in one breath To indicate the type of case in which a diagnosis be-
een an embolus from a myocardial infarct and a thrombus of a stenosing
opliteal atheroma is hardly possible, one may cite the case of myocardial
farction in a 65 year old man who was severely hypotensive for three days
ter a coronary occlusion On the fifth day, an acute arterial occlusion of the
moropopliteal segment occurred Extraction of the clot eight hours later
vealed severe popliteal atheromatosis The thrombus reformed and the
itient lost his leg The thrombosis undoubtedly occurred in the stenotic
gment during the long period of hypotension and was not an embolus

The site of the occlusion has, of course, a great deal of influence on the
itcome of the operation. Statistics would indicate that embolectomies on
e upper extremity are overwhelmingly successful On my own service we
ive not found it necessary to operate on upper extremity arterial occlusions,
it one would not hesitate to do so if a preliminary paravertebral block
iled to rewarm the extremity In the past, the results of popliteal embolec-
my and especially of thrombectomy were the poorest I agree with Warren
id his associates[117b] that popliteal occlusions did fairly well with conserva-
ve treatment Later a sympathectomy can improve collateral circulation
ie recent aggressive approach of Shaw[118] and Crawford and DeBakey,[119]
ho flush out a soft thrombus from the posterior tibial artery into the
opliteal or femoral artery with the help of a 50 cc syringe, is a marked
lvance in the surgical treatment of emboli It only fails if there is a previ-
isly occluded segment in the lower leg, or if the musculature has become
reversibly necrotic I have used a ureteral catheter for this purpose, the
trograde flushing is much better

All embolectomies are comparatively minor procedures and done under
cal anesthesia, with the exception of the saddle embolus of the aorta This
quires a laparotomy under spinal anesthesia If cardiac reserve is poor or
the patient is in rapid fibrillation or congestive heart failure, the aortic
nbolus should be attacked through the two femoral arteries under local
iesthesia. This is usually not as satisfactory, but I have seen patients sur-
ve with viable limbs The mortality of a saddle embolus of the aorta is very
gh in any case An ill timed laparotomy adds to this figure

(6) The last procedure which may become necessary after an acute
terial occlusion is *amputation* Factors which operate in this unfortunate
itcome are (1) the site of the occlusion, (2) the time elapsed between the
cclusion and admission to the hospital, (3) the cardiac status and peripheral
rculatory failure, (4) ill advised measures of local heat, ice packs or mas-

sage and (5) lack of protection of the ischemic extremity with large cotton wraps

When the extremity is obviously lost amputation should be urged When permission is obtained the extremity is packed in ice with the help of six ice bags which are renewed every four hours Waiting for demarcation is a peculiar desire to postpone the inevitable and as is sufficiently known may lead to higher amputation toxemia, longer duration of phantom pain and loss of life

Chronic Arterial Thromboses

These will be handled under the underlying diseases which they accompany and complicate Both thromboangiitis obliterans and arteriosclerosis obliterans are invariably accompanied by thrombosis A monograph by Edwards especially stresses the point that the arteriosclerotic individual escapes severe disability or gangrene until thrombosis occurs in the diseased vessel [120]

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CHAPTER 13

VENOUS INSUFFICIENCY (VARICOSE VEINS) OF THE LOWER EXTREMITIES

VENOUS INSUFFICIENCY OF THE LOWER EXTREMITIES IS A FREQUENT AND HIGHLY disabling condition, its management has undergone a great number of variations since Hippocrates (500 B C) punctured a varicose vein Superimposed on the nature of the malady and its progressive course are ill advised or ill timed procedures which greatly add to the chronic disability of the general population and lead to an economic strain on the patient and his supporting community.

CLASSIFICATION OF VARICOSE VEINS

It is customary to speak of primary and secondary varicosities, the first being due to a congenital or acquired weakness of the venous wall or its valves, and the second being the consequence of a deep venous occlusion by thrombosis, which is followed by a collateral circulation to compensate for the obstruction Practical considerations, however, indicate that this differentiation can often not be made and that the question which really arises is. what is the immediate cause of the venous hypertension in the erect position, and where are the valves whose insufficiency is directly responsible for the presence of the obvious dilatation of the superficial venous system The valvular incompetence may be in the long or short, in the communicating or in the deep venous systems The recognition of these varieties rests on an adequate case history, but mainly on a simple examination of the state of venous return On this examination rests the proper method of treatment (fig 203)

THE ETIOLOGY OF VARICOSITIES

The congenital type of varicosities is not uncommon, and we have been especially impressed by a group of patients in whom the venous backflow develops early and becomes manifest around puberty Such patients often

show a pluriglandular disturbance with a deficiency of their pituitary corticoadrenal axis poor resistance to stress low basal metabolic rates obesity and lack of elasticity of their supporting structures Their adaptation to posture is poor They show a fall in blood pressure in the erect position partly because of their low vasomotor tone and partly because of the pooling of 500 to 1,000 cc of blood in their lower extremities when they stand They later develop multiple venous thrombi especially after pregnancy (fig. 204) The valves here are incompetent in all three systems and cosmetic results after treatment are often poor Not to be confused with this group are patients with congenital arteriovenous communications since their management is entirely different The latter exhibit cutaneous birthmarks and an overgrowth of the limb with increased temperature and blood of arterial color can be aspirated from the presenting venous dilatations (see chapter 7 Congenital Vascular Anomalies)

Varicosities following trauma are frequent but the history of a subsequent phlebitis is not always obtainable Sprained ankles contused calves and minor or major injuries to the lower extremities may suggest little involvement of the superficial communicating or deep veins at the time of the injury Weeks or months later a characteristic pattern of collateral veins appear these veins are not necessarily connected with the long saphenous system and show direct connections with deep veins through incompetent perforators

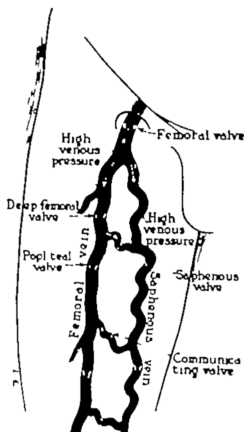


FIG 203 The valvular defect may be in the superficial, perforator or deep venous system. In this diagram all three systems are incompetent. Sudden rises in venous pressure, such as occur on straining, sneezing or coughing, send waves of impulses into a system where pressure is high on standing and does not fall on exercise.

What has been said about varicosities following trauma is especially true of those following operations and childbirth The damage to the deep and perforator veins by thrombosis, recanalization and valvular incompetence may be subclinical, but the pattern of the veins and the tests directed toward the site of valvular leaks leave no doubt that an old deep venous thrombosis occurred at the time of operation or delivery

Much has been written about the varicose veins of pregnant women They have always been treated as if the pregnancy were not present, up to the seventh month, after this only elastic supports are used and surgical treatment postponed to three months after delivery It is more than likely that in addition to the mechanical interference with venous dra nage by the enlarging uterus and the increased vascularity of the pelvis, a hormonal effect is present Attention has been called to the numerous painful intradermal clumps or bluish vessels which become hot and tender and which improve on the administration of estrogen and/or progesterone [1] We have no personal experience with this type of therapy.

Chronic infections or vascular accidents requiring prolonged bedrest, such as pneumonia or coronary thrombosis, are frequently the cause of varicosities, again through the agency of a recognized or unrecognized deep venous occlusion resulting in collateral circulation



FIG 204 An obese woman in her early thirties Always a fat child, she started to menstruate at the age of 15, had scant irregular menses, and had left deep thrombophlebitis with each of three pregnancies Note the malleolar and genicular fat pads and bursae



FIG. 205 Extensive phlebosclerosis with calcification in a long saphenous vein. The patient's arteries showed no clinical or roentgenological evidence of sclerosis.

Malignancy either by directly compressing large venous trunks in the pelvis or by causing thrombosis through a hypercoagulability of the blood and prolonged recumbency often manifests itself by the appearance of superficial varicosities

The traumatic postoperative postpartum postinfectious and malignant types of varicosities are all post thrombotic There remains a final variety the phlebosclerotic one to which little attention has been paid in the past. In this the femoral and popliteal veins undergo a vascular sclerosis analogous to arteriosclerosis with resulting dilatation tortuosity and valvular shrinkage Such a vein equal to the post thrombotic recanalized vein allows gravitational backflow from above and leads to deep valvular insufficiency [2] It is amazing how seldom this occurs in the saphenous system (fig. 205)

TESTS FOR VALVULAR INCOMPETENCE

The test for valvular insufficiency of the long saphenous vein is the Trendelenburg test (fig 206) It is performed by raising the patient's lower extremity well above the horizontal level, then placing a rubber band or a finger around the root of the limb and asking the patient to stand up suddenly Sufficient light must be present to enable the examiner to watch for the rapidity with which the visible varicosities refill in the erect position

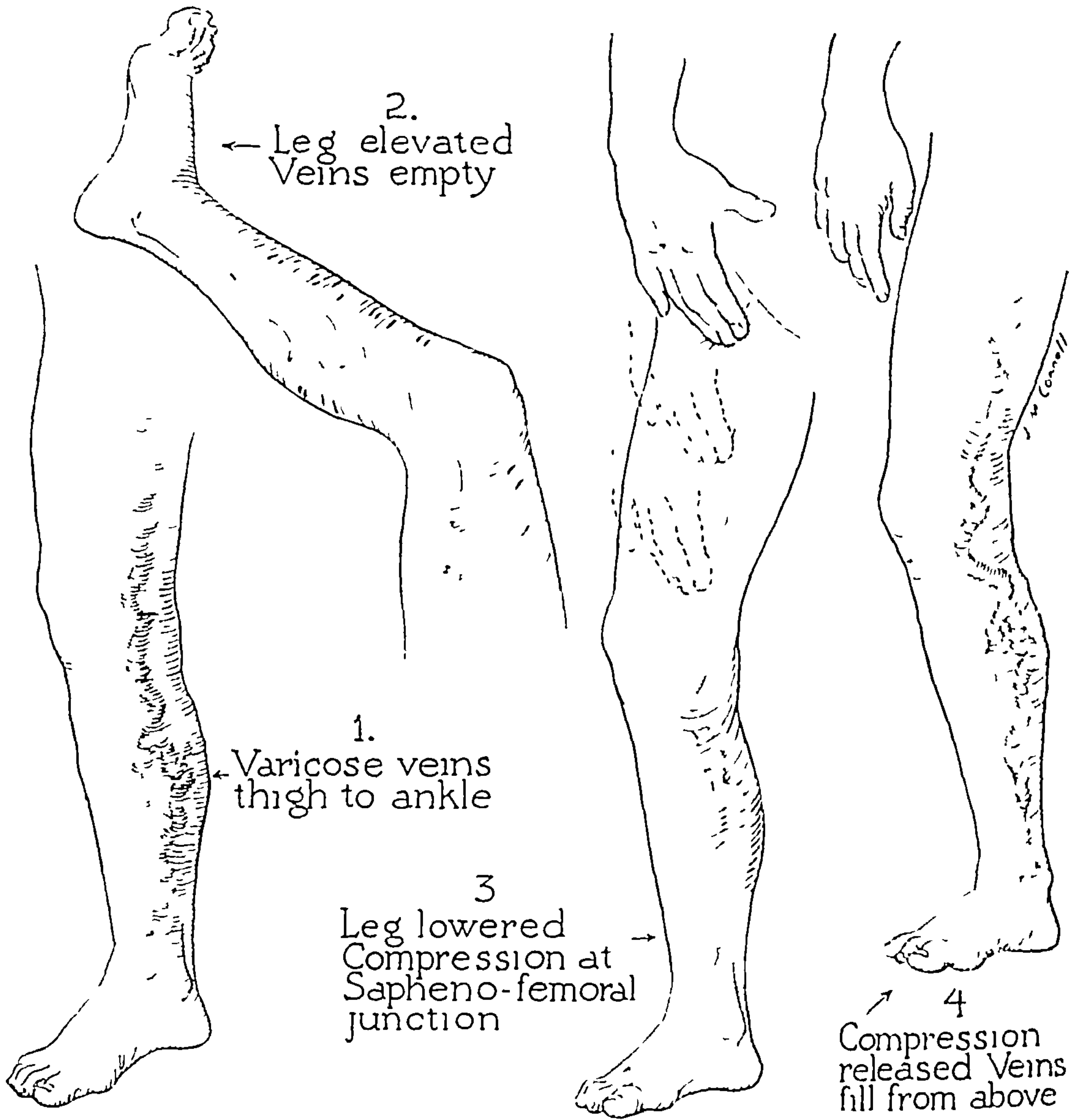


FIG 206 The classic Trendelenburg test It really picks out the cases of isolated long saphenous valvular incompetence, which give excellent prognosis but constitute the minority of varicosities seen

They may stay empty, they may refill partially or they may suddenly and completely distend On releasing the tourniquet, the amount of reflux from above can be readily estimated If the veins stay empty while constriction was maintained, the valvular incompetence is mostly in the long saphenous vein If the veins do not or only partially collapse on standing with the band in place, the flow of blood must come from an overflow through incompetent perforators The level of these perforators may be roughly studied by lowering the level of the tourniquet to midthigh or by using different levels of digital compression above and just below the knee, thus allowing an estimate of the site of incompetence Recent studies of Sherman,[3] however, have shown the multiple and complicated pathways through which blood can regurgitate from the deep to the superficial system, therefore too much reliance can not be placed on these segmental Trendelenburg tests

If the veins have promptly and completely refilled in the standing position in spite of elastic compression above the site of varicosities one has to resort to some form of muscular exercise to see whether or not the veins can be emptied or at least partially collapsed by the muscle pump This is the principle of the Perthes test and its modifications One test which we have recently described helps to differentiate between deep venous obstruction and deep venous insufficiency The patient first elevates and lowers his affected extremity as in the Trendelenburg test except that the tourniquet is not at the groin but below the knee (fig 207) If this measure flattens his veins there can be neither serious deep venous insufficiency nor obstruction resulting in overflow from incompetent perforators If the veins remain distended the patient is made to stand on his toes ten times rapidly with the tourniquet in position The veins may remain distended in spite of this exercise indicating deep venous obstruction they may disappear on this exercise indicating deep venous patency or they may disappear and refill on standing still with the tourniquet in place indicating deep venous insufficiency Obviously the obstruction patency or insufficiency does not relate to a single vein but to the sum total of the deep venous system One can supplement or confirm the findings of this simple clinical test by measurements of venous pressure (fig. 208) or by visualization of the deep venous system (fig. 209) For the overwhelming majority of patients however these additional tests which require trained personnel and much time are not necessary



FIG 207 Test for deep venous insufficiency or obstruction. The patient first elevates his leg well above horizontal to empty all varicosities A rubber band is now placed snugly below the knee and he suddenly stands up In (a) the veins in the calf remain collapsed the patient's deep and perforator valves are holding. Should the veins remain distended, he stands on his toes rapidly ten times as in (b) The veins remaining distended now indicate deep venous obstruction. The veins disappearing on exercise indicate deep venous patency as in (c) The veins disappearing on exercise but refilling again on standing still with the tourniquet in place as in (d), indicates deep venous insufficiency or regurgitation

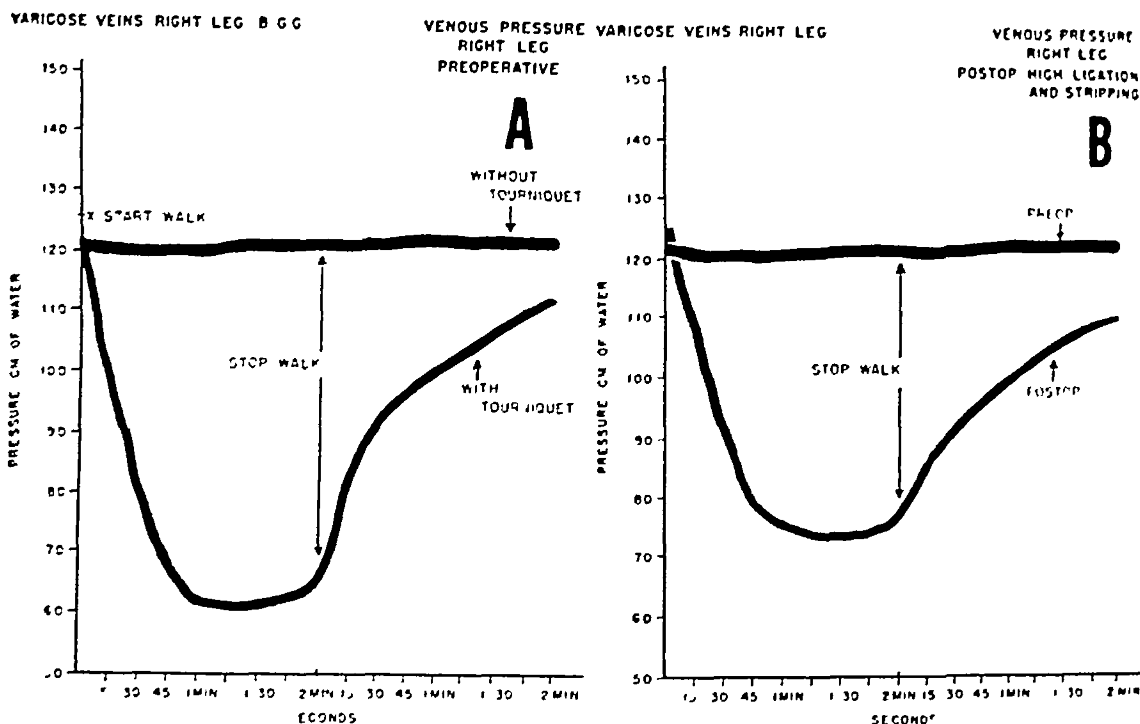


FIG 208 Walking venous pressure measurements of the right leg (A) Before the operation the venous pressure is 122 cm H_2O while patient stood or walked With a tourniquet on, the pressure drops to one half of the standing pressure on walking, which is the normal response and predicts good result from vein ligation and stripping (B) After the operation, pressures drop to a low level on walking without the tourniquet (de Takats, G Postphlebetic Syndrome J A M A , *164* 1861, 1957 Courtesy of Dr John H Schneewind, U of Illinois College of Medicine)

THE INTERPRETATION OF SYMPTOMS

Patients affected with varicose veins of the lower extremities complain of unsightly spiders, bulbous dilatations, swelling, a tired, heavy feeling or a bursting pain on standing, all of which may be readily explained by the elevated venous pressure in the erect position Greatest caution must be exerted, however, in interpreting a variety of symptoms as being connected with the presence of varicosities From flatfoot to arthritis of the small joints and ankles, from muscle hernias to arthritis or internal derangement of the knee joint, from chronic inguinal lymphadenitis to arthritis of the spine, slipped disk and spondylolisthesis, from saphenous neuritis caused by a thrombophlebitic exudate in the femoral sheath to alcoholic polyneuritis, a multitude of conditions should be thought of and excluded before attributing the patient's complaints to obvious varicosities

Varicose veins, unless they are visibly inflamed, do not hurt day and night They do not cause cramping on walking unless arterial insufficiency supervenes They do not produce a sciatic type of pain on raising the leg, they do not manifest themselves by shooting radiation along the lateral cutaneous nerve and they do not cause an absence of ankle jerks in diabetics All this is stressed to indicate the necessity of a thorough examination and proper interpretation of the patient's symptoms The number of patients

with vein ligations and stripping procedures whose symptoms continue or are aggravated by insufficient indication for surgery is considerable

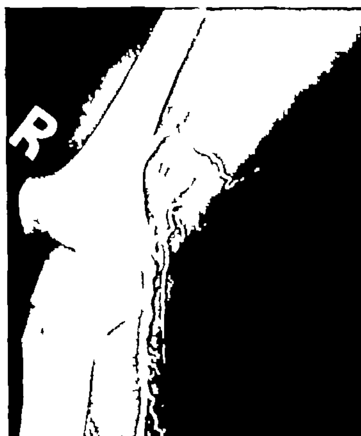


FIG 209 Visualization of the popliteofemoral venous system with 35 per cent Diodrast solution shows patency and normal contour of this segment Below the knee, however the contour of the posterior tibial vein is ragged, the opacity uneven and collaterals are numerous. A limited deep venous thrombosis exists below the knee with recanalization. (de Takats, G and Fowler E. F Varicose Veins of the Lower Extremities. Surgical Clinics of North America, *31* 1463 1951)

TREATMENT

There are three phases in the surgical management of varicosities which have been practiced in our clinic In the late 1920 s the ambulatory ligation of the long saphenous vein was started in the groin and followed by injection.[4] The emphasis was on the regurgitation of blood at the saphenofemoral junction and an effort was made to ligate all tributaries at this level (fig. 210) This is still an integral part of the procedure and ligations made too low and encouraging the development of inguinal tributaries are seen all too often Religation after insufficient low ligations must be made with an effort to get above the scarred area and above hyperplastic lymph glands (fig. 211)

The high ligation was then followed by biweekly or weekly injections of sclerosing solutions, a whole number of agents being used which now have

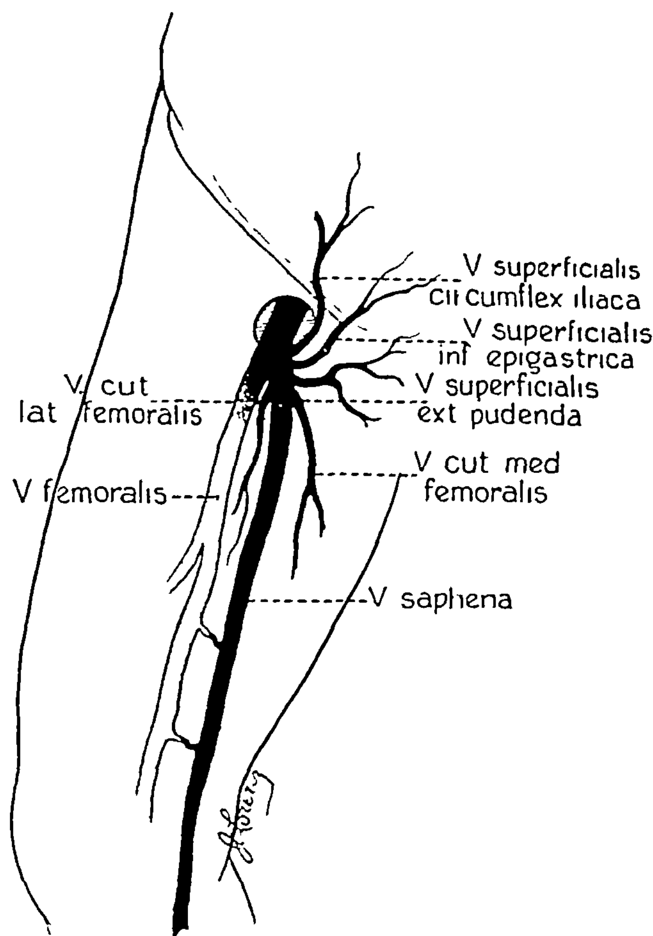


FIG 210 The saphenofemoral junction with a pattern of tributaries which are far from being constant The division of the vein must occur flush with the femoral vein

only historical interest, such as hypertonic solutions of sodium chloride or dextrose, sodium salicylate, quinine and urea, sodium morrhuate and potassium oleate As pointed out in earlier writings, such solutions must be nontoxic, injectable in small quantities through a hypodermic needle, not too irritating and not allergenic In the last few years, sodium tetradecyl sulfate (Sotradecol) in 1 and 3 per cent concentration in doses of ½ to 1 cc at each location has been the only sclerosing solution used. Since the drug produces very little periphlebitic reaction, four to six injections may be done in one sitting

The injections themselves need minimal equipment The patient's veins in the standing position are first marked out with a drop of tincture of iodine He then lies down and a blood pressure cuff is placed on his thigh and inflated to 60 mm Hg to distend the vein A 2 cc syringe armed with a 25 gauge needle aspirates the sclerosing solution from a rubber-capped ampule After a clean venipuncture, free blood must flow from the vein on aspiration. The solution can now be injected into the distended vein if it is small, if the varicosity is large, the rubber cuff can be deflated so as to inject a half empty vein, thus minimizing the ensuing thrombus The index finger of the left hand is kept over the venous bulge to make sure that no perivenous injection is made If the airblock or airfoam technique of Orbach[5] is used, the ballooning

of the skin may herald a paravenous injection but I have not employed this technique Actually it is very true that small amounts of air often get mixed with the sclerosing solution and this helps to clear the intima of blood for the sclerosing solution and does not lead to any symptom of air embolism even after 15 000 injections done with the airblock technique [6] A small strip of adhesive tape with sterile gauze in the center (a Band Aid) is placed on the site of puncture It is advisable to bandage the leg following multiple injections since this reduces the pressure in the dilated veins and hastens a firm obliteration without bulky thrombosis



FIG 211 Ligation of the long saphenous vein is too low here to include all tributaries. There is a visible and palpable bulge in the groin just below Poupart s ligament The recurrence seen here occurred two years after an insufficient ligation The line of incision and the visible tributaries are marked with a dye. Such cases require religation (Christopher F A Textbook of Surgery)

The number of injections was formidable in the early days and was obviously necessitated by the recanalization of thrombi when incompetent perforators fed blood under high pressure into the superficial system The percentage of recurrence after this combination of high long saphenous ligation followed by injections was high in some reports as high as 50 to 90 per cent (in literature cited by Orbach[5]) While some authors who use nothing but sclerosing injections alone have claimed far better end results our group became dissatisfied with injections alone, with injections preceded by high ligation and with high ligation followed by retrograde injections, which in

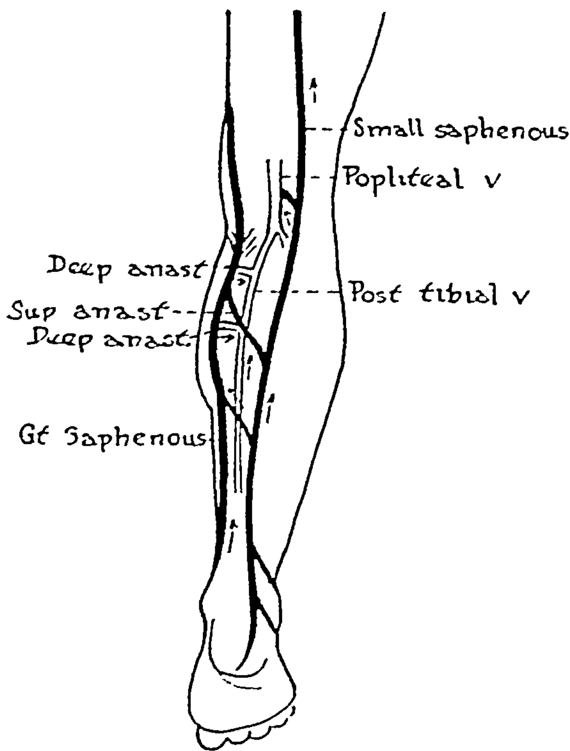


FIG 212



FIG. 213

FIG 212 The long course of the short (small) saphenous vein anastomosing freely with the long (great) saphenous vein and also with the popliteal (de Takats and Quint Surg Gynec. and Obst, 50:549, 1930 After Kosinski)

FIG. 213. Localized phlebectasia in the short saphenous system After ligation such a vascular mass is excised and not injected since it would cause bulky clots Stripping of such tortuous veins is impossible but by finger dissection they can be straightened out and extracted by multiple, small incisions

one particular group of 367 extremities only resulted in anatomical and symptomatic relief for 55 per cent of the cases [7]

In looking for the causes of failure in the treatment for varicose veins, it was pointed out in 1931[8] that the reflux of blood into the dilated saphenous varicosities may occur from other sources than the saphenofemoral junction More attention was now paid to the short saphenous vein in the popliteal fossa, with its developmental anomalies studied by Kosinski [9] He pointed out that the short saphenous vein emptied into the popliteal vein in only 57 3 per cent of the cases, terminated high on the thigh emptying into the long saphenous vein in 12 per cent of the cases, entered both the popliteal vein and the deep veins of the thigh in 27 per cent of the cases and ended low in the upper third of the calf in 9 7 per cent of the cases (fig 212) Kosinski pointed out in 1926 that the short course of the short saphenous vein in man is probably an adaptation to posture and that the persistence of a long course of this vein, either emptying into the femoral vein or into the long saphenous vein on the thigh, may explain some of the extensive tortuous varicosities in the popliteal fossa (fig 213) While statistics based on observations in patients are always subject to the special interest of the examiner, we can say that roughly 10 per cent of the patients have a manifest involvement of the short saphenous system [10] Since our clinic has noticed an increase of short

saphenous valvular incompetence after long saphenous ligation and stripping which contributes to recurrence it is wise to look for small bulbs at or around the popliteal fossa or below it If the vein is enlarged and tortuous at the external malleolus, it is advisable to divide it and strip it centrally to eliminate early involvement which may later become troublesome With this principle in operation roughly 50 per cent of our patients operated on in the last few years for varicose veins and 100 per cent of those operated on for post phlebitic varicose veins were subjected to short saphenous ligation and stripping.

Stripping itself an old procedure was carried out as early as 1930 with both a Babcock and later a Mayo stripper when too many incompetent per forator valves were thought to be present [11] However stripping was done only occasionally in 1930 radical excision of the main trunk from groin to ankle having been practiced in 16 out of 60 patients reported by Quint and myself [12] Looking back on this procedure today it was not only unneces sarily traumatic but led to many recurrences because the most important perforators were missed

A great impetus for the reawakening of interest in the stripping procedure came from Thomas T Myers who not only developed an excellent flexible stripper but who reported on 2 660 stripping operations and 510 secondary operations for persistent or recurrent varicosities [13] Following his lead a number of other strippers have been devised I have had experience with only one other namely that of Emerson and Muller [14] This stripper consists of a flexible cable with a cone shaped flange on either end which in turn is threaded for a short woven silk bougie It proved to be too delicate for the rigors of an active vascular service since the tips were bent broken or lost in the shuffle The Myers stripper if handled with some care by the house staff and nurses, has stood the strain better The Zollinger stripper is cer tainly rugged enough but seems to be more traumatizing than that of Myers It is serviceable on large thickened, periphlebitic veins

It became obvious, however that stripping the two main channels namely the long and short saphenous veins did not eliminate the incom petent perforators which often connected with the main trunks and con tinued to feed the high pressure into superficial varicosities Quint and I referred to this source of retrograde filling in 1930 [12] but perforator in competence was a comparatively infrequent problem at that time since patients with a history of deep thrombophlebitis or with a typical pattern of collateral varicosities were not considered suitable for surgical procedures

The Committee on Scientific Exhibits of the American Medical Associa tion prepared varicose vein exhibits at the Annual Scientific Sessions pam phlets were issued at each of these meetings and revised in 1931 1941 and 1944 In the last pamphlet,[15] multiple ligations were advised, combined with injections at the level of perforators However many of the patients now accepted for surgery were rejected because while stenosis and regurgitation were recognized as being the result of recanalized deep and perforating vein thrombosis by Edwards and Edwards,[16] most such patients were diagnosed

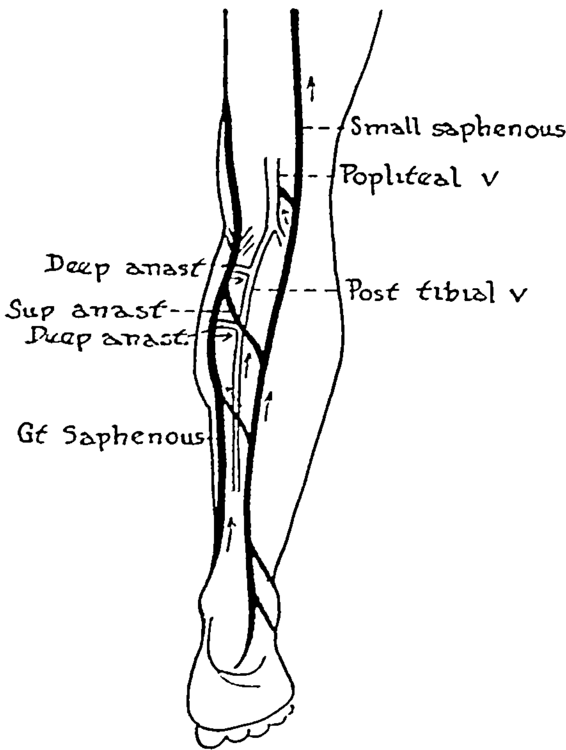


FIG. 212



FIG 213

FIG 212 The long course of the short (small) saphenous vein anastomosing freely with the long (great) saphenous vein and also with the popliteal (de Takats and Quint Surg Gynec and Obst, *50* 549, 1930 After Kosinski)

FIG. 213. Localized phlebectasia in the short saphenous system After ligation such a vascular mass is excised and not injected since it would cause bulky clots Stripping of such tortuous veins is impossible but by finger dissection they can be straightened out and extracted by multiple, small incisions

one particular group of 367 extremities only resulted in anatomical and symptomatic relief for 55 per cent of the cases [7]

In looking for the causes of failure in the treatment for varicose veins, it was pointed out in 1931 [8] that the reflux of blood into the dilated saphenous varicosities may occur from other sources than the saphenofemoral junction More attention was now paid to the short saphenous vein in the popliteal fossa, with its developmental anomalies studied by Kosinski [9] He pointed out that the short saphenous vein emptied into the popliteal vein in only 57.3 per cent of the cases, terminated high on the thigh emptying into the long saphenous vein in 12 per cent of the cases, entered both the popliteal vein and the deep veins of the thigh in 27 per cent of the cases and ended low in the upper third of the calf in 9 7 per cent of the cases (fig 212) Kosinski pointed out in 1926 that the short course of the short saphenous vein in man is probably an adaptation to posture and that the persistence of a long course of this vein, either emptying into the femoral vein or into the long saphenous vein on the thigh, may explain some of the extensive tortuous varicosities in the popliteal fossa (fig 213) While statistics based on observations in patients are always subject to the special interest of the examiner, we can say that roughly 10 per cent of the patients have a manifest involvement of the short saphenous system [10] Since our clinic has noticed an increase of short

saphenous valvular incompetence after long saphenous ligation and stripping which contributes to recurrence it is wise to look for small bulbs at or around the popliteal fossa or below it If the vein is enlarged and tortuous at the external malleolus it is advisable to divide it and strip it centrally to eliminate early involvement which may later become troublesome With this principle in operation roughly 50 per cent of our patients operated on in the last few years for varicose veins and 100 per cent of those operated on for post phlebitic varicose veins were subjected to short saphenous ligation and stripping.

Stripping itself an old procedure, was carried out as early as 1930 with both a Babcock and later a Mayo stripper when too many incompetent per forator valves were thought to be present [11] However stripping was done only occasionally in 1930 radical excision of the main trunk from groin to ankle having been practiced in 16 out of 60 patients reported by Quint and myself [12] Looking back on this procedure today it was not only unneces sarily traumatic but led to many recurrences because the most important perforators were missed

A great impetus for the reawakening of interest in the stripping procedure came from Thomas T Myers who not only developed an excellent flexible stripper but who reported on 2,660 stripping operations and 510 secondary operations for persistent or recurrent varicosities [13] Following his lead a number of other strippers have been devised I have had experience with only one other namely that of Emerson and Muller [14] This stripper consists of a flexible cable with a cone shaped flange on either end which in turn is threaded for a short woven silk bougie It proved to be too delicate for the rigors of an active vascular service since the tips were bent broken or lost in the shuffle The Myers stripper if handled with some care by the house staff and nurses, has stood the strain better The Zollinger stripper is cer tainly rugged enough but seems to be more traumatizing than that of Myers It is serviceable on large thickened, periphlebitic veins

It became obvious however that stripping the two main channels namely the long and short saphenous veins did not eliminate the incom petent perforators which often connected with the main trunks and con tinued to feed the high pressure into superficial varicosities Quint and I referred to this source of retrograde filling in 1930 [12] but perforator in competence was a comparatively infrequent problem at that time since patients with a history of deep thrombophlebitis or with a typical pattern of collateral varicosities were not considered suitable for surgical procedures

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as having a lack of patency in the deep veins and a latent infection in the superficial ones [17] Thus they were eliminated both from ligations and from injections and were relegated to continuous elastic support It is interesting to note that some clinics still adhere to this principle today and many patients are encountered who have been told "never to have their veins removed or stripped because of their stopped-up deep circulation "

The changed attitude of operating on patients who have definite history or evidence of deep venous thrombosis really started with the aggressive attitude of the Vascular Clinic of the Massachusetts General Hospital under the leadership of Robert Linton [18] He systemically exposed a medial, lateral and anterior group of perforators, ligating them under the fascia I have found this operation most helpful in the multiple perforator incompetence of the medial perforators, especially when heavy postphlebitic induration with lymph stasis makes the skin vulnerable and does not permit extensive undermining or suprafascial ligation Linton[19] also advocated the interruption of the superficial femoral vein for the postphlebitic syndrome, in the belief that massive reflux through the recanalized superficial vein was responsible for the venous stasis—and all its complications—in the erect position This principle of attacking the valvular incompetence of the recanalized deep venous system was supported by Bauer, who has carried out the interruption at the popliteal level In his latest article[20] he reported on the division of the popliteal vein in 650 patients together with resection of all accessory popliteal veins After a three year follow-up, 75 per cent of the patients were asymptomatic with healed ulcers and in 25 per cent either edema or ulceration recurred once or more often

We ourselves reported on a small series of popliteal vein ligations[21] for the bursting pain, edema and ulceration of the postphlebitic leg. While we were impressed with the early results, a few years ago we abandoned all deep venous ligations for the postphlebitic syndrome While it is true that the increased venous pressure is aided in the erect position by deep venous ligations, during walking or exercise the venous pressures are not diminished but are increased [22] In our institution, John Schneewind has had identical experience [23] Significantly, the University of Michigan group, after an early favorable report and an average follow-up period of eight years on 45 patients, has come to the conclusion that deep vein ligation in the postphlebitic extremity did not produce any significant improvement in symptoms [24]

It is believed, therefore, that while an aggressive attack on the venous system crippled by incompetent valves of the deep and perforating veins is justifiable, this attack should be limited to the incompetent perforators, even though it is clear that their incompetence is only secondary to high postural pressure in the deep veins One is then in a third era of surgical treatment of varicose veins, the first era was the high ligation of the saphenous, the second was the recognition of ligation and stripping of the long and short saphenous veins, and the third era is the localization and surgical interruption of incompetent perforator veins

The first significant contribution here was the painstaking and revealing

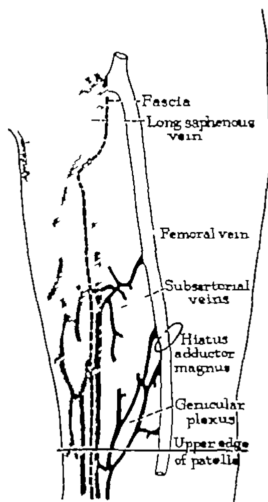


FIG 214 The diagram, modified and simplified from the illustrations of Sherman depicts a heavy trunk of the long saphenous system dipping under the fascia *at various levels* It shows the mid Hunterian the subsartorial and the genicular veins which are constant, and when incompetent need interruption. (Sherman B S Varicose Veins. Ann. Surg. *120* 772, 1944)

study of Sherman [3] While he first concentrated his efforts on the perforator system of the thigh[3] (fig. 214) it became clear that the incompetent leg perforators were really the crux of the problem since these were more numerous and were much more often responsible for incomplete therapy Sherman especially emphasized a mid Hunter canal perforator system, the genicular plexus perforators and accessory perforator veins in the leg, which are not obliterated by the stripping of the long or short saphenous vein

Incompetent perforator veins are very common in the leg Any attempt to systemize them in a diagram is bound to fail, both because of individual variations in anatomy and chiefly because not all of them become incompetent and need division. However Dodd and Cockett in their excellent monograph[25] have given some of the typical locations of these perforators and have re-emphasized the fact that the most important ones on the medical surface of the leg are a good inch posterior to the path of the long saphenous vein The outward flow from deep vein to superficial at a characteristic location above the inner malleolus is shown in figure 215

In the foregoing, a historical development of the surgery of varicose veins has been presented with the obvious implication that the majority of varicosities seen are not, as had been originally supposed due to an isolated long saphenous vein involvement with all valves functioning except those of the long saphenous vein (fig. 216) In fact, out of 100 consecutive cases 60



FIG 217 (a) A gumma, (b) postphlebitic induration with multiple communicating valvular incompetence, (c) bilateral postphlebitic lymphedema, (d) arteriosclerotic ulcer, (e) traumatic, infected ulcer with osteomyelitis, (f) postphlebitic ulcer in a fractured limb with additional arteriosclerosis, (g) erythema induratum (Bazins disease)

which has been hit on the tibia In (e) there is a large traumatic ulcer over an old fracture of the tibia, a type of case which requires saucerization of the bone and expert plastic surgery, possibly a skin flap for closure In (f) is a postphlebitic ulcer, associated with an old fracture and much venous stasis as a result of deep venous thrombosis occurring at the time of fracture In (g) are multiple tuberculous ulcers, an erythema induratum, which is a dermatologic problem and not a vascular one

The object, of course, is to heal the ulcer, and adept bandaging has to be taught When the lesion is depressed in the hollow of the ankle, foam rubber placed over fine mesh gauze aids in producing adequate compression and also has protective value Salves with a petrolatum base, which does not mix with the serous or purulent drainage, do more harm than good They macerate the skin and prevent absorption of drainage by the gauze A water-soluble base or jelly is preferable, but we only use it as a solvent for streptokinase-streptodornase (Varidase) powder, which is our preferred preparation to dissolve necrotic or fibrinous exudates If necrotic eschars

need this enzymatic treatment, they should be scarified and frequently gently loosened so that the enzyme can get into contact with the necrotic material

Probably the best method of treating inflamed postphlebitic ulcers is absolute bedrest elevation of the bed on 8 inch shock blocks, hot fomentations, unless they are contraindicated by ischemia or by wet dermatitis and saturated boric acid dressings applied to the granulating surface unless it is necrotic When half strength Dakin s solution is used with two Dakin tubes the skin around the ulcer needs protection with Lassar s paste

It is obvious however that elevation mild heat and keeping the ever menacing *Bacillus pyocyaneus* away with boric acid or ¼ per cent phosphoric acid solution are only preliminary measures toward definitive treatment

If the ulcer is small the induration slight or minimal and the varicosities markedly incompetent, the previously described multiple ligation and stripping procedure is indicated Should one have to get *under the ulcer* to ligate the incompetent perforators, it is wise to place the incision posterior to the ulcer toward but not over the Achilles tendon and preferably through normal skin Such a situation is shown in figure 218 in the case of Mrs F F who 12 years after the operation shows no edema and no recurrence of ulceration but only a pigmented atrophic skin over the inner malleolus In another case operated on in 1940 (fig 219) whom we had recently had the opportunity to re-examine, the incision was placed too close to the tibia and left a sensitive scar Some of the missed perforators are seen posteriorly in the upper third of the leg The two ulcers however were sufficiently undercut and stayed healed for 16 years

In another group of patients after cooling off the acutely inflamed ulcer



FIG 218 Firmly healed postphlebitic ulcer in Mrs. F F., 70 years old who 12 years previously had had the ulcer undercut and multiple perforators ligated She wears a light nylon elastic hose. There has been no recurrence of ulceration or edema. Note the position of incision avoiding the pigmented atrophic skin Grafting here was not necessary

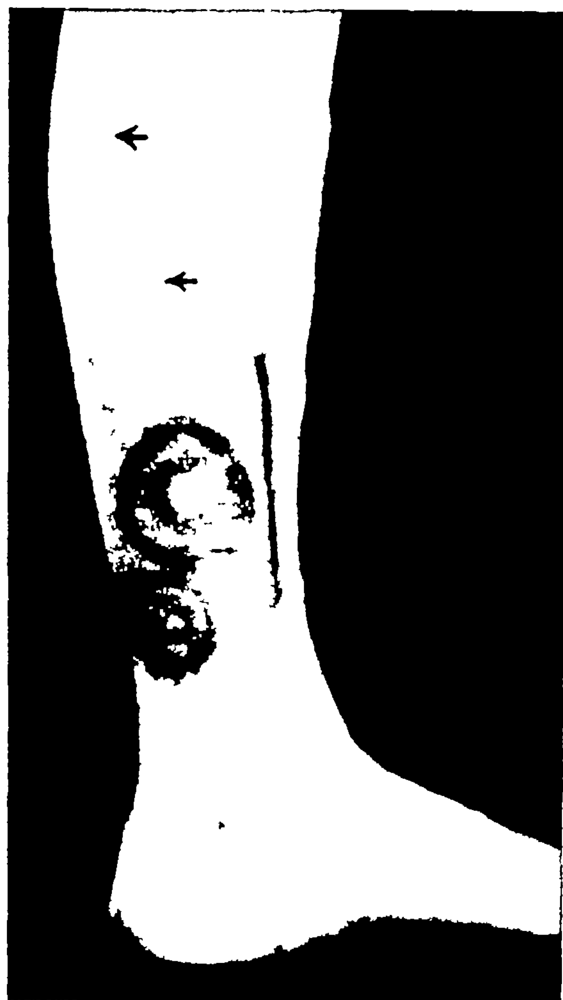


FIG 219 In 1940, two resistant posttraumatic and postphlebitic ulcers were undercut with a medial incision The bases of these ulcers were lifted from the fascia, thereby sectioning the perforators, but no individual ligations were done The ulcers remained healed for 16 years The line of incision and the healed areas are marked with Mercurochrome Note the tortuous, short to long saphenous communication in the upper third of the calf

it is possible to apply an Unna's boot which is changed from one to three weeks, depending on the amount of secretion Unna's paste, consisting of 100 Gm of zinc oxide, 100 Gm of gelatin, 100 Gm of glycerin and 200 Gm of water, is best prepared by the hospital druggist,* although impregnated bandages in cans ready for use are obtainable, they are inferior to the heated ones

The leg is painted from toe to knee with the paste which has just been melted in the water bath A 3 inch gauze bandage is wound over the leg, avoiding all folds This paste is also useful in the postoperative period following extensive stripping and multiple ligation of perforators, since it provides a semielastic hygroscopic dressing and is well tolerated, unless the patient is sensitive to zinc oxide, which is very rare Its use, however, is contraindicated in copiously secreting ulcers, in patients with an acute dermatitis and in hot weather, when it will ooze and melt into the stocking

The local use of antibiotics and also of Furacin (5-nitro-2-furaldehyde semicarbazone) produces such a high percentage of sensitization that one

* The zinc oxide is mixed with enough water to make a thick paste, and the glycerin is added while the mixture is being stirred The gelatin is softened in cold water and the water is squeezed out as quickly as possible The gelatin is then heated on a water bath and, while it is being constantly stirred the zinc oxide and glycerin mixture is added to the solution When the paste has cooled, it is cut into cakes weighing 8 oz each

cannot advocate their use Because of the local venous and lymph stasis, anti bodies seem to be concentrated in this area. Again and again one can observe autosensitization meaning that the patient becomes sensitive to his own secretion, breaks out in a rash around the ulcer and later breaks out in a generalized rash This seems to occur especially when occlusive dressings with waxed paper or adhesive tape are used and we therefore are in favor of letting the drainage be absorbed on the dressing It is easy to teach the pa tient to change his own dressing, cleanse the lesion with a hexachlorophene (pHisoHex) preparation and place dry preferably fine mesh gauze over the ulcer followed by snug bandaging with an elastic porous bandage (fig 220) When there is no draining area other forms of semirigid containment * notably a 4 inch impregnated starch bandage which is porous and sticks to itself but not to skin (Gauztex) may be used As pointed out by Freeman and Coelho the muscle pump can squeeze tissue fluid more effectively against a rigid than against an elastic dressing Yet this material is porous and pliable and when pressure points such as the heel the dorsum of the foot, the ankles and the tibial tuberosity are padded with felt it can be worn without abrasion or blistering Especially after operations for lymphedema this dressing has been found to be very useful one police officer puts it on himself changing the dressing every four weeks

The use of the muscle pump in squeezing edema out of the limb is naturally limited when the edema is hard with much fibrosis to prevent mobilization and when the main venous channel is blocked, since the increased tissue pressure and venous pressure can then be not relieved (fig. 221) For

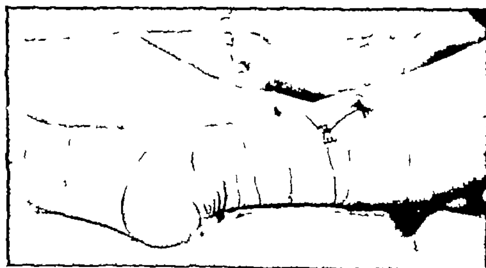


FIG 220 Customary but improper application of elastic bandage. The bandage should be wider (4 inches) the heel should be included and the bandage should extend to the tibial tuberosity Note the swelling in the submalleolar area due to improper bandaging. (de Takats, G and Fowler E. F Varicose Veins of the Lower Extremities. Surgical Clinics of North America, *31* 1463 1951)

* This term and the ideas expressed here regarding the impregnated, starched gauze are those of Norman E. Freeman of San Francisco who together with Helio M Coelho has developed this method.

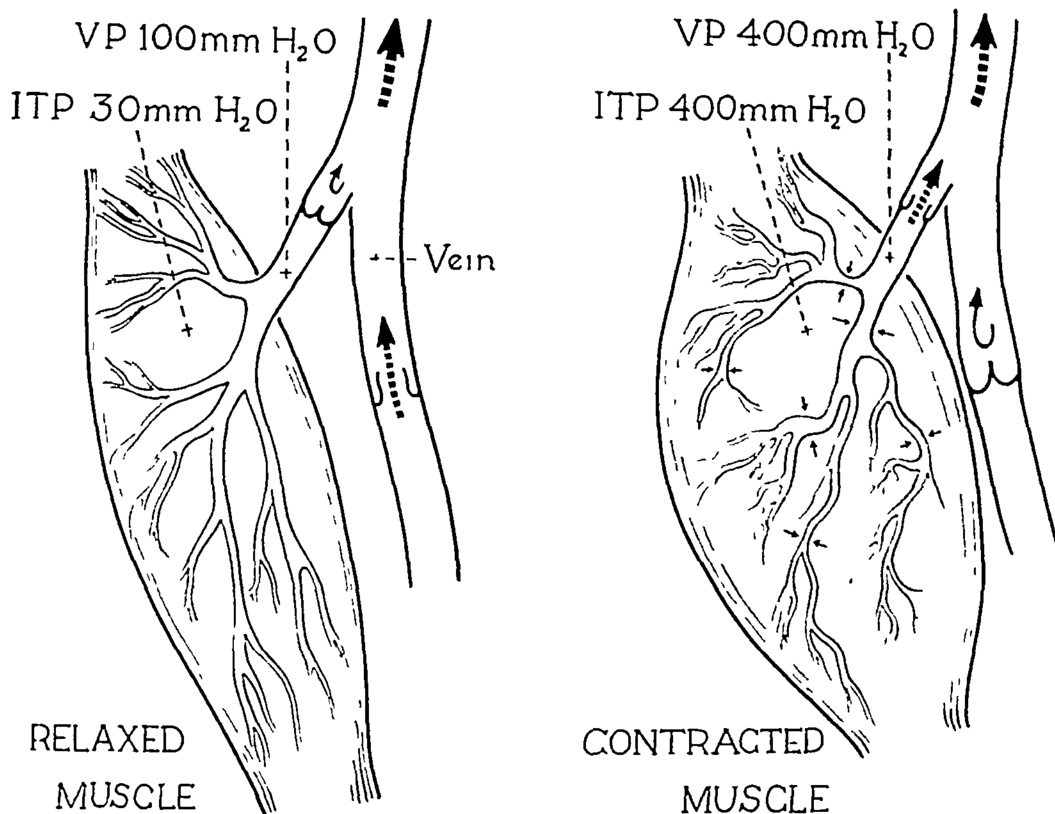


FIG 221 Intramuscular tissue pressure (I T P) is relatively low when the muscle is relaxed, but increases considerably when the muscle contracts The rise in venous pressure (V P) will be high in case of deep venous obstruction or on *standing* with deep venous insufficiency (Burch, G E A Primer of Venous Pressure Lea and Febiger, Philadelphia, 1950)

this reason, an acute deep venous obstruction seldom tolerates exercise with or without outside compression In the chronic phase, most obstructions are greatly relieved on walking Standing still or sitting still in cars, trains, planes, air raid shelters and in front of television sets not only increases edema but may bring on venous thrombosis

The second surgical method, used to treat postphlebitic induration without ulcer, is to incise the area from the level of the ankle to its proximal extent, prepare the flaps of skin with a thin layer of subcutaneous tissue and excise the entire area of fat necrosis, fibrosis, thickened lymphatic trunks and perforators down to the fascia When the fascia is thickened, it too is excised, thus performing a micro-Kondoleon operation The success or failure of this method will depend entirely on the state of the pigmented atrophic skin with diminished or absent capillary and lymphatic circulation Tests for the circulation of the postphlebitic skin consist in using histamine flares[27] described in part II, Methods of Diagnosis, and in using sky-blue dye to delineate the area devoid of lymphatic circulation [28] In the past, it has been possible in many instances to save the skin overlying the induration, but recent experience indicates a disconcerting percentage of sloughs following radical excision of the induration The cause of the slough is not only the poor character of the skin, but, as pointed out by John Homans many years ago,[29] the lack of adequate venous drainage, even though stab wounds for drainage and adequate compression are always used For this reason, the

conclusion has been gradually reached that unless the incision for ligating the perforators and excising the induration can be placed *outside* the visibly involved skin the skin had better be excised with the underlying tissue

Here again two methods are available for closing the ensuing defect In large and previously infected ulcers it is best to excise them and wait for healthy red buds of granulations to appear before a split thickness graft is placed This, of course means a week or two of delay less important in the charity ward than in a private hospital Or one can immediately cover the defect and with some exceptions this has been our method of choice The skin is usually taken from the same side and the use of a Furacin soluble dressing with fine mesh gauze at the donor site may cut down delayed healing usually due to secondary infection [30] The percentage of sensitization here is low about 2 per cent much lower than if the drug had been applied to the ulcerated area where we never use it The skin itself is removed from the same thigh with the Padgett or Brown electric dermatome. Thicker than 012 to 015 inch grafts usually heal poorly although I have occasionally taken a free full thickness graft this was done with notable success on frost bitten Marines with gangrenous patches on their heels or fingertips In this area, however and because the size of the grafts is considerable, the split thickness graft is the only dependable one

A 100 per cent take may occur but should not be counted on in the ch onically infected area One can count on the healing of both the donor area and the host area in three to six weeks (fig 222)

Grafts usually shrink, but if placed on too loosely may wrinkle and infection or small ulcerations may develop in their hillocks and crevices The graft shown in figure 223 was placed in 1932 and photographed in 1954 22 years later It belongs to a beauty parlor operator who stands consistently 8 to 10 hours while at work. She has faithfully worn an elastic bandage and has had no ulceration in the graft When the graft is sutured under too much tension there is marginal necrosis In fact some surgeons will place no sutures at all into the graft This has also been done on our service and is a rapid, convenient method, but it is my impression that the graft needs a small amount of tension, so that when perforated with a number 11 blade for drainage the small stab wounds will remain open

The grafts stand up under stress remarkably well provided all saphenous and perforator valvular incompetence is eradicated and an elastic bandage is worn to contain edema. The graft will break down on (1) direct injury to the graft, (2) persistent swelling due to deep venous insufficiency inade quately supported or (3) arterial or arteriolar damage Even so in a 57 year old diabetic obese cook with a low I Q who only intermittently bandaged her leg, a graft stayed in place for 10 years (fig. 224)

Generally speaking, a patient with a split thickness graft done for recur rent postphlebitic ulceration and induration, needs to bandage her leg during the day for the rest of her life If she develops an inverted bowling pin deformity owing to scar contraction and fibrosis (fig. 225) a foam rubber pad must fill out the hollow above the inner malleolus Out of 200 split

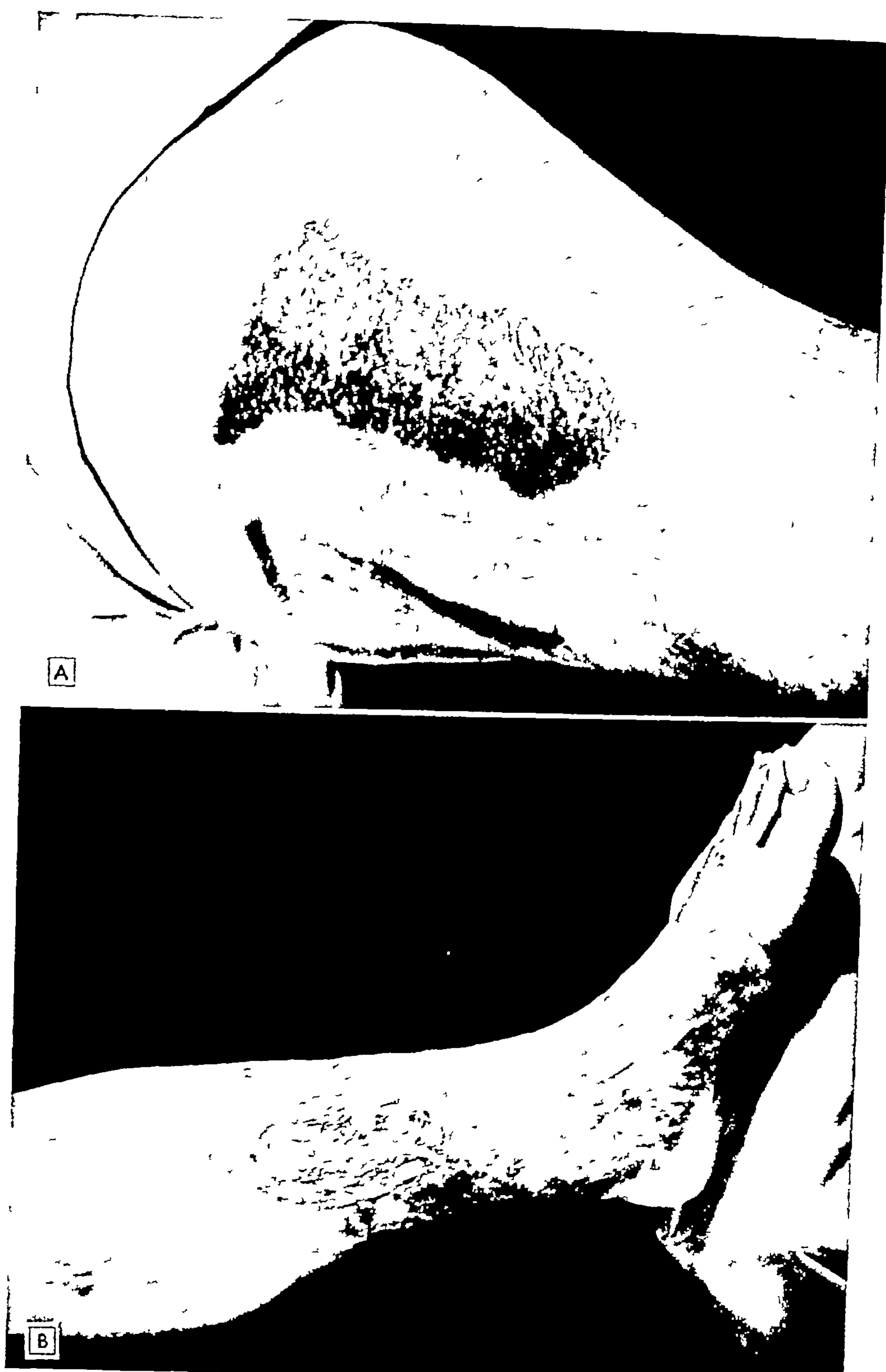


FIG 222 (A) Donor area dressed with a Furacin soluble dressing three weeks after the operation Note that only part of the drum was functioning This may be due to the cement or in this instance to an *eccentric drum* (B) Site of the graft three weeks after the operation on the same patient The graft remains flaky and dry for a long time, although axons will grow into it Sweat gland function may return partially, but is not normal 50 per cent alcohol in glycerin is a useful tanner and lubricant.

thickness grafts followed carefully from 1 to 20 years 21 broke down for one reason or another Five of these have been successfully regrafted [31]

A number of clinics have advocated lumbar sympathectomies in the case of the postphlebitic syndrome We have very seldom performed this



FIG 223 A 22 year old skin graft in Betty R a beauty parlor operator The graft is uneven and there is fibrous tissue proliferation under it, but it has stayed unbroken in spite of the patient s strenuous occupation.



FIG 224 Split thickness graft in an obese diabetic cook in the neighborhood. The graft stayed in place for 10 years. There is no ankle edema. Bandaging was very intermittent

operation, only using it when there was associated vasospasm, a minor causalgia with postphlebitic neuralgia or excessive hyperhidrosis to indicate the usefulness of this procedure

As pointed out in a recent article on hyperhidrosis,[32] a postphlebitic ulcer surrounded by dermatitis may evoke a reflex type of sweating limited only to one extremity In addition, there is vasospasm and the foot and leg are cold, purplish and numb Such extremities are moist, dripping wet and the seat of continuous or recurrent skin infection and an intractable derma-



FIG 225 Marked retraction of fibrous tissue above both malleoli of the right leg, giving the appearance of an inverted bowling pin Ligation and stripping of the long and short saphenous veins and ligation of incompetent perforators had been combined with excision of the induration and a split thickness graft The leg can not be well supported without filling out the supramalleolar hollow with foam rubber

titis (fig 226) Preceding the skin graft it is wise to perform a lumbar sympathectomy to abolish hyperhidrosis, it has helped the wet dermatitis and the recurrent fungus infections of such patients

But it should be emphasized that these are rare cases, together with the postphlebitic causalgias, and that the overwhelming majority of patients suffering from the postphlebitic syndrome need no sympathectomy Not only is it unnecessary, but as pointed out by Robert Linton[18] a "post-thrombotic postsympathectomy syndrome" develops, characterized by chronic, painful ulcerations on the dorsum of the foot and on the toes, in contradistinction to the original lesion above the inner ankle There is increased edema and while



FIG 226 A huge postphlebitic ulcer in a 30 year old woman following a postpartum phlebitis. There are beads of perspiration in the upper left quadrant distal to the ulcer The heel is to the left of the picture. (de Takats, G The Surgical Treatment of Hyperhidrosis. Arch. Dermat and Syph., *76* 31 1957)

arterial inflow is increased venous pressure is higher and ambulation increases the swelling. While such cases do occur we have never done any sympathectomies for edema or ulceration unless either reflex sympathetic phenomena or an arterial impairment would justify it and this syndrome has not been encountered

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CHAPTER 14

LYMPHEDEMA

LYMPHEDEMA RESULTS FROM OBSTRUCTION TO OR REFLUX OF LYMPH CAUSING retention of tissue fluid with consecutive fibroplasia, induration of fat thickening of fascia and enlargement of the involved area. Lymphedema, how ever may also be due to maldevelopment of the lymphatics The morbid anatomy revealed by lymphangiography is different in these two types [1] Only lymphedema of the extremities will be considered here

ANATOMICAL CONSIDERATIONS

The lymphatic system of the extremities starts in the tissue spaces drained by lymph capillaries which in turn converge into collecting channels trunks and lymph glands (fig. 227) The *lymph capillaries form a closed net* work, making hollow projections into the papillae of the skin The lymph vessels draining the lymphatic network are colorless ½ to 1 mm in diameter and are beaded due to numerous valves One can differentiate superficial from deep lymph vessels separated by the fascial layer covering the muscle mass Generally speaking, they accompany veins and drain the correspond ing territories although the lymph vessels are more plentiful than the veins

The lymph vessels traverse one or several groups of lymph glands which vary in size from a pin s head to an olive unless they are pathologically en

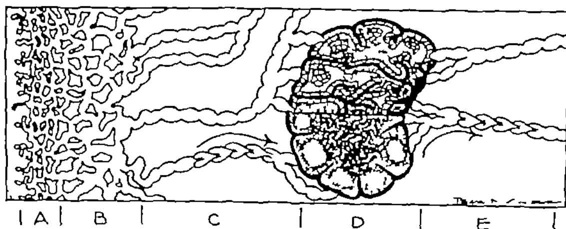


FIG 227 The structural basis of the lymphatic system (semidiagrammatic) (A) The irregular blindly-ending lymph capillaries (B) the reticular network (C) the afferent tubular lymphatics, containing valves (D) the lymph node, presenting above the lymph sinuses and below the histologic picture (E) the efferent lymph vessels, showing numerous valves above the arrow (Curtis in Christopher F Textbook of Surgery)

larged. Lymph is brought to the sinuses of the cortex by several channels and then leaves by vessels arising in the medulla.

When small particulate matter is injected into an afferent lymph channel, a blockade may be produced in the cortex of the regional lymph gland. This results in a retrograde flow in the lymph channel to the nearest anastomosing lymph trunk, which affords a detour around the blocked lymph gland into one lateral to it or even distal to it. The whole situation is analogous to the development of collateral circulation after a venous block. Furthermore, if the lymph clots, which is enhanced by the products of inflammation, the valves are destroyed and one is faced then with a retrograde lymph flow in the dependent position of the extremity.

THE MECHANISM OF LYMPH RETENTION

Lymph itself is a clear, colorless fluid with a strikingly different concentration of protein when the extremity is at rest compared to when it is active. Drinker[2] gave figures of 1.8 to 2.28 per cent of protein in the lymph of an animal at rest and between 0.5 and 1.5 per cent when the same animal was walking about. In experimentally produced thrombophlebitic edema, Zimmerman and I[3] obtained figures of tissue fluid protein as high as 3.4 per cent.

In an ascending lymphangitis with enlargement of the regional lymph nodes, permanent obstruction to the channels and lymph sinuses may occur. One encounters this after erysipelas, after an infection of the fingers or toes and frequently after a fungus infection with secondary invasion of bacteria. This is an *ascending* type of blockade. Conversely, a lymph block may occur following extirpation of regional lymph nodes or malignant invasion of them, producing secondary *descending* or retrograde type of lymphatic obstruction. It would seem that the extent of the peripheral involvement of lymphatics, especially their closure by inflammatory exudate and the clotting of lymph, will determine how irreversible the edema is.

When capillary permeability increases because of injury, venous stasis or superimposed inflammation, a protein-rich, interstitial fluid appears, which has difficulty in being transported away by venous or lymphatic drainage. Zimmerman and I showed in 1931[3] that this protein-rich exudate precipitates and forms a fibrinous network. This "plasma clot" also operates in burned areas[4] and in immersion limbs.[5]

The fibrinous exudate acts as a scaffold for the proliferation of fibroblasts and forms a hard, irreversible, nonabsorbable lymphedema. Clark and Clark[6] found that this exudate contains growth-promoting substances and also a fibrinolytic ferment which dissolves the fibrin and causes marked retraction of tissue. This is precisely what is seen in an old postphlebitic induration due to lymph stasis, which retracts and gives a peculiar inverted bowling-pin appearance to such affected lower legs. Repeated small traumatic insults or infections produce bouts of inflammation, which in turn are responsible for larger and harder patches of fibrosis. The growth-promoting

substance or substances are probably akin to those operating in the healing of wounds, and a discussion of these would take us far afield It should be emphasized however that early drainage of tissue fluid either by adequate transport through lymph channels or by drainage to the outside markedly diminishes the residual fibrosis Thus accumulation of plasma in a poorly drained amputation stump will lead to bulky hard and painful overgrowths of fibrous tissue

The obliterative lymphangitis caused by ascending infection particulate matter or an overload by fibrinous exudate, reaches the lymph nodes and plugs their sinuses Obliterative streptococcus lymphangitis is a recognized entity but attention has been called by Valy Menkin to the exotoxin of *Staphylococcus aureus* which produces necrotizing material and lymphatic blockade [7]

Large pools of tissue fluid develop which are undrained and the valvular apparatus of the lymphatic system is thrown out of commission The superficial lymphatic system in the corium contains very few valves to start with [8] and when a dye is injected into this system it will drift about in any or all directions governed by gravity or movement of the part, as soon as there is any obstruction [9]

Obviously retained lymph is the imbalance between increased secretion and inadequate removal of tissue fluid The studies of McMaster have clarified many of the factors which govern interstitial fluid pressure and resistance [10] In clinical cases it is important to determine whether or not an increase in lymph production by producing venous hyperemia, by exercise or by massage will improve or aggravate lymphedema

The studies of Kinmonth and his co-workers[1] indicate however that congenital, juvenile and even some of the traumatic and inflammatory lymphedemas are caused by an absence or poor development of lymphatics and the obvious causes simply precipitate a decompensation of lymphatic circulation

TYPES OF LYMPHEDEMA

In 1950 Evoy and I[11] reported on 150 personally observed cases of lymphedema and in tabulating them used a simple classification which avoids such nomenclature as lymphedema praecox or Milroy s disease (Table X) The same classification is followed here

Congenital Lymphedema

The edema, together with port wine stains hemangiomas and lymphangiomas appears at birth but may not be noticed by the parents until some time later We have encountered lymph cysts, half solid half fluctuating masses in all soft tissue planes of the extremities and also in the retroperitoneal space for which reason this area is routinely explored when surgical treatment is begun

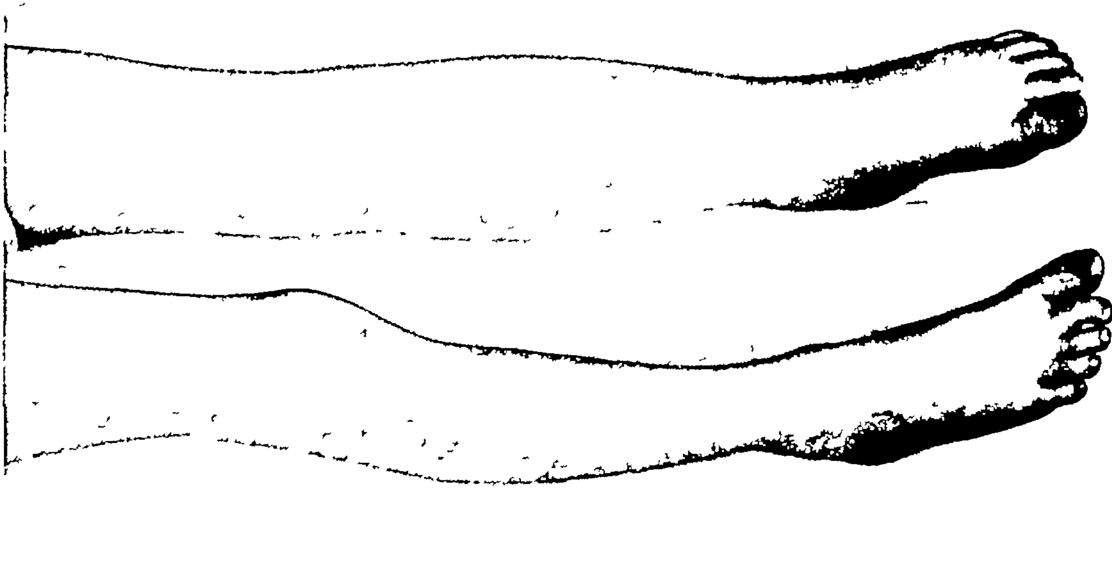


FIG 228 Unilateral lymphedema in David L , a 6 year old boy

Often there are no associated birthmarks and certainly no infectious etiology For the 6 year old boy in figure 228, who is now 13, four operations have been performed and his edema is under fair control He is not behind in his studies and leads a normal life

A congenital lymphatic-vascular anomaly is shown in figure 229 Multiple stage incisions revealed lymph cysts together with large vascular dilatations

The combination of lymphatic and vascular anomalies may be so extensive, however, that amputation and sometimes hemipelvectomy may be suggested to avoid much suffering and mental stress to the child and parents For the 6 month old baby, in figure 230 a hemipelvectomy was advised, although understandably refused Hemipelvectomy was successfully performed in a mixed hemolymphangioma on a 13 day old child [12]

Table X

LYMPHEDEMA IN 150 PATIENTS

ETIOLOGY	NUMBER OF PATIENTS
Congenital	28
Traumatic (includes axillary and inguinal dissections)	11
Inflammatory	58
Degenerative (malignant)	22
Unknown	31
	150

35 males 23%
115 females 77%



FIG 229



FIG 230

FIG 229 Lymphatic vascular anomaly in a 3 year old girl. Lymph cysts and angiomatous masses were present

FIG 230 Lymphatic vascular anomaly in Laura L. 6 months old Hemipelvectomy was refused Her further course is unknown.

The upper extremity together with the chest wall is frequently involved In the case of Mary S an ingratiating lady of 1 year the chest wall on the affected side was also involved and we have seen this to occur in other cases (fig 231)

In the case of Bruce B a 3 year old boy, two stages of excision have been completed The enormous cushion on his left hand is seen before operation in figure 232 A

There is no inflammatory component in this lesion and the tissues may remain soft and pitting it is not true in our experience that the difference between venous and lymphatic edema is that of consistency Lymphedema does become hard when repeated attacks of infection supervene so does venous edema when a lymphangitic (periphlebitic) component enters into the clinical picture

It is customary to speak of Milroy s disease when the congenital lymphedema is hereditary but realizing how many ancestors of patients have thrombophlebitic, cardiac or nephrotic edema great care must be exercised in diagnosing such a case Among more than 150 patients seen with lymph edema, we have had not a single case in which parents or grandparents could be examined and found to have true lymphedema



FIG 231 Lymphedema of the chest wall and left upper extremity in a 1 year old girl



A



B

FIG 232 Lymphedema in Bruce B, a 3 year old boy Operations were started on the hand in (B)

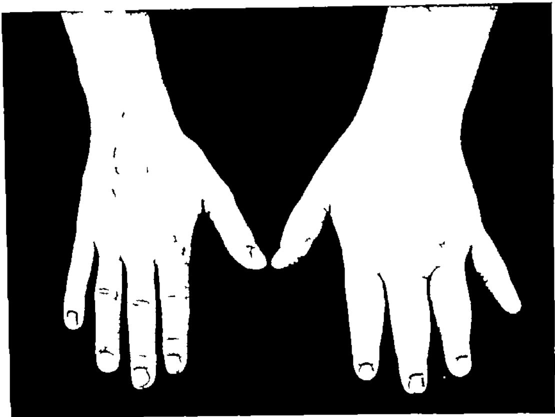


FIG 233 For a fractured metacarpal bone a circular cast was applied, which was later bivalved. This soldier developed a massive hard edema over the dorsum of the hand without any vasomotor symptoms or osteoporosis. The function of the hand was permanently impaired.

Traumatic Lymphedema

Every soft tissue injury results in changes in capillary permeability and accumulation of tissue fluid This *acute lymphedema* may be aggravated by such factors as hemorrhage a tight bandage a cast, a dependent position and secondary infection Unless this acute edema is relieved by simple measures a permanent thrombosis of the lymphatics and a fibrosis of the tissues may result with impairment of function (fig. 233)

Causalgic states are often accompanied by a chronic lymphedema which is thus of traumatic origin The retention of lymph flow here is partly due to a complete immobility of the injured part which the patient holds in a fixed and often dependent position There may be a vasomotor element in the edema since it is reduced after sympathetic block with the relief of pain but even before the resumption of muscle activity Figure 234 shows the right arm and hand of a 16 year old school girl who has held this extremity in a fixed and dependent position for several months following an insignificant trauma to her shoulder Such causalgic edemas later develop into shrunken, atrophic glossy hands with irreversible subcutaneous fibrosis Ill advised splints and casts and prolonged immobilization often add to the difficulty

Injury to regional lymph nodes in the axilla or groin following dissection may lead to lymphedema Holman McSwain and Beal[13] believe that a com



FIG. 234 The right upper extremity of this patient is edematous from fingertips to shoulders in a causalgic state several months after an automobile accident

bination of infection and radiotherapy is responsible for the lymphedema of the arm following radical removal of the breast Actually, clean dissection of the axillary lymph nodes does not have to result in lymphedema of the arm either because a few nodes are always left intact or because of the well known regenerative powers of the lymphatics [14]

Postmastectomy lymphedema is one of the major causes of physical and psychological handicap following radical mastectomy (fig 235) It is difficult to get exact figures on its incidence, but it probably occurs in *all cases* for a short time Venous obstruction because of axillary vein removal or secondary thrombosis is certainly not a major factor Secondary infection of lymphatic extravasations and postmastectomy radiation are potent factors Prophylaxis of such lymphedema consists in adequate drainage of the axillary space, with continuous suction if necessary, and avoidance of pressure dressings to the shoulder area.

Following the injury to lymph nodes, especially if they are enlarged and partially obstructed, a *lymphorrhea* may develop which may last for weeks and months unless checked early with 150 to 200 roentgen units

Such lymphorrhea has been encountered after incisions in the groin, at the ankle and at the knee Certainly one should handle lymph glands with extreme care and gently retract them out of the field instead of cutting across

them or across their afferent or efferent channels The secretion has a peculiar characteristic odor it is crystal-clear and seems to have an inhibiting action on granulations Since Carlson and Luckhardt s classic review on ferments in lymph (1908–1909) [15] not much has been added to our knowledge on the chemistry of lymph from the extremities because most studies refer to lymph in the thoracic duct.[16] Clinically the atonic pale granulations and the sweetish odor of clear fluid exuding from the neighborhood of enlarged lymph channels or glands is characteristic of lymphorrhea



FIG 235 Postmastectomy lymphedema, one and one half years following radical mastectomy This arm was heavy but not painful.

Inflammatory Lymphedema

When bacterial or perhaps viral infection gets into the lymphatics a spreading obliterative lymphangitis develops Ochsner and his associates discussed in detail the importance of reinfection and local tissue sensitivity in the production of recurrent attacks [17] Attempts to find the offending organism are frequently unsuccessful Blood cultures during or within a few hours after the chill of recurrent lymphangitis are usually negative Only in three patients out of a great many in whom cultures were attempted on our service was the culture positive A green producing streptococcus was found in a 10 day blood culture in a second patient, an acute tonsillitis which yielded a *Streptococcus viridans* seemed to have coincided with an acute

flare-up In an 18 year old boy with a congenital lymphatic-vascular anomaly, chills and fever with the erysipeloid rash was followed within the hour by a positive blood culture, diagnosed as *Streptococcus zymogenes.* For the success of the blood culture, it is important that antibiotics be withheld until the blood sample has been obtained and that the patient or his family be alerted to the importance of obtaining the blood at or shortly after the chill The positive blood culture then supplies the source for an autogenous vaccine, the use of which will be discussed on page 373

Clinically, one can readily recognize a tubular type of lymphangitis by the *red streak* of periphlebitis involving the collecting lymphatics and ascending to a regional lymph node The nodes enlarge and become painful The *reticular* type, on the other hand, produces the red, hot, raised patches of skin Single attacks of tubular or reticular lymphangitis seldom if ever lead to persistant lymphedema Only if the lymphangitis recurs, if it involves a large group of lymph glands, or, chiefly, if the early attacks are ignored or ill treated, will the edema remain permanent

The port of entry of the offending organism is frequently at the fissures or blisters between the toes Trichophytosis is known to be a frequent cause of scales and blisters and the secondary invader is most often the streptococcus Often the lymphedema is limited to the toes and foot and can be stopped from ascending by adequate therapy Whether the fungi or the secondary invaders are primarily responsible for tissue sensitization has been debated, vaccine therapy of trichomoniasis has been suggested [18]

In addition to the toes, a source of lymphatic invasion through the vagina has been suggested in a study undertaken with Evoy [11] Vaginitis, cervicitis and pelvic lymphadenitis certainly occur frequently without causing any lymphedema of the lower extremity However, the pelvic or presacral nodes may spread a lymphatic blockade in a retrograde fashion to the inguinal nodes, and vaginitis and cervicitis drain toward the inguinal lymph glands The collecting lymphatics from the vagina and cervix form a paracervical plexus, which in turn drains into the external iliac, the internal iliac and the presacral nodes (fig 236)

Hyperplasia of regional lymph nodes occurs not only as a direct spread of infection but also as an immunological reaction, since the lymph follicles produce antibodies The lymphatic channels also serve to localize an infection to the site of invasion by becoming plugged with thrombi or a fibrinous exudate As Valy Menkin pointed out,[19] the gauge of the invasiveness of an organism depends on the rapidity and intensity with which a lymphatic blockade is established Thus, staphylococci usually localize readily because they isolate themselves with the rapid production of a lymphatic blockade On the contrary, the hemolytic streptococcus produces a relatively mild local reaction and does not close the lymphatics for as long as two days

I have gone into the mechanism of blockade in some detail because of the therapeutic implications of early, intensive treatment Whether or not a lymphedema will remain irreversible will depend on the lymph nodes' ability to clear lymphatic pathways and sinusoids from fibrin and cellular debris,

at the same time protecting the body from a systemic invasion of pathogenic bacteria Most of the patients with lymphedema are seen at a late stage, with secondary infections repeated bouts of chills and fever trichophytosis and trichomonas vaginalis Because of the appearance of such a limb the patient usually a woman is depressed despondent and overeats, and rehabilitation is difficult (fig 237) Episodes of chills and fever as many as three to four a year keep increasing the lymphatic obstruction The process is often activated by a fungus infection with a delayed reaction to inflammation (chapter 9 Arterial Inflammation) (fig 238)

The histologic picture of lymph glands removed at inguinal biopsies is usually that of an nonspecific lymphadenitis The periadenitis which is noticed at taking the biopsies strangles the active hyperplastic follicles and prevents entrance and exit to the gland (figs 239 240) Inguinal biopsies may reveal an unsuspected Hodgkin s disease as in the case of a young cashier in whom general lymphatic spread and death followed several years later or they may reveal a tuberculous lymphadenitis as in the case of a middle aged undernourished Negro man who showed no pulmonary tuberculosis But

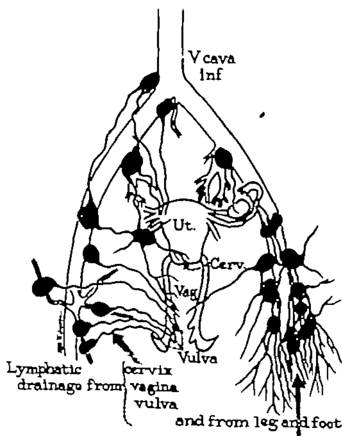


FIG 236. Diagram of the lymphatic apparatus draining the inguinal, iliac and aortic nodes. On the left, drainage from the cervix, vagina and vulva toward the iliofemoral bottle neck is shown on the right, the lymphatic drainage from the lower extremity is illustrated. Drainage from the parametrium is indicated toward the iliac, sacral and retroperitoneal nodes which surround the vena cava and aorta. In case of lymphatic obstruction at the nodes, retrograde and collateral flow of lymph occurs. (de Takats and Evoy Lymphedema. Angiology *1* 73 1950)



FIG 237 Inflammatory lymphedema in Mary C , a 45 year old housewife, whose edema started with a stormy, febrile course with chills The leg was never bandaged Seven years before this picture was taken, a Kondoleon operation was performed Note the raised cutaneous warts and the "pigskin" of chronic cutaneous lymphstasis Bedrest, mercurial diuretics, autogenous streptococcus vaccine and adequate elastic support permit her to perform her duties as a mother of nine children (de Takats and Evoy Lymphedema Angiology, *1* 73, 1950)

generally speaking, the histologic picture reveals little, although no lymphedema of the lower extremities should be treated without a preliminary biopsy The finding of an unsuspected malignancy will be discussed below

The lymphedematous tissue itself goes through stages of progressive fibrosis and dilatation of lymph channels The corium thickens, the collagen fibers are elongated and thickened and there are large lymph spaces in the subcutaneous tissue in which the lymph moves to and fro by gravity (fig 241) In addition to the enlargement of lymph channels, the increased tissue pressure causes arteriolar thickening and collapse of venules, as shown in the center of figure 242 Finally, there develops an irreversible fibrosis with broken-up elastic fibers which can never function, so that even if the mobilizable fluid is evacuated, the skin and connective tissue will not contract around it (fig 243)

Malignant Lymphedema

Glandular metastases to regional lymph glands not only block lymphatic circulation but may produce retrograde implantation of tumor cells We have observed striking examples of this, such as a patch of carcinomatous skin on the heel of a patient with cancer of the uterus, pigskin of the upper extremities in carcinoma of the breast, and a lymphedematous and carcino-

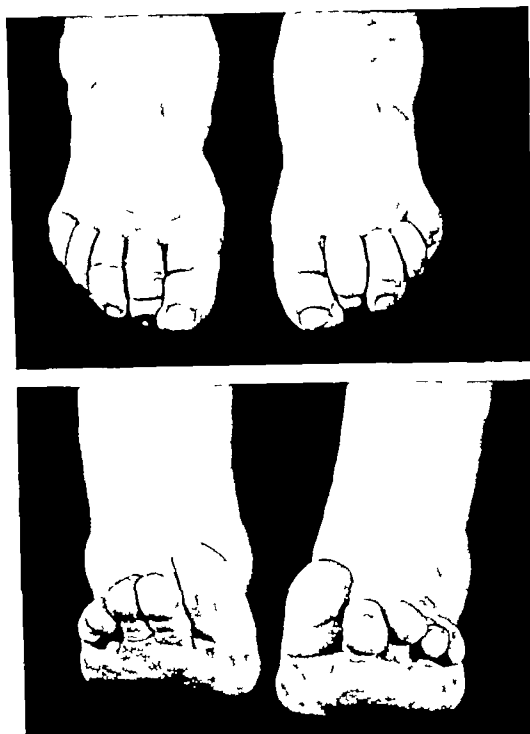


FIG 238 R.S a 45 year old man, with inflammatory lymphedema of both lower extremities. Note the fissures and ulcerations between the toes caused by trichophytosis. The toes are lymphedematous, which is not the case in venous edema.

matous penis following carcinoma of the bladder Attention has also been called to the occurrence of lymphosarcoma in lymphedematous extremities of nonmalignant origin not unlike Kaposi s hemorrhagic sarcoma [20] In every case of slowly progressive lymphedema without obvious congenital traumatic or inflammatory etiology the possibility of lymphatic block due to gland metastasis or a primary malignant involvement of lymph nodes due to lym-



FIG 239 Inguinal lymph node taken from E E, a 37 year old woman with bilateral progressive lymphedema She had a history of three febrile miscarriages Note the active lymph follicles and the marked periadenitis at the bottom and right of the photomicrogram A pelvic lymphadenitis with a retrograde spread to the inguinal lymph glands was diagnosed



FIG 240 Inguinal lymph node of Isabel E, a 42 year old woman Note the almost complete disappearance of lymphoid tissue, replaced by fibrous and hyaline masses This gland is functionless Enlarged lymph spaces are seen in the left border of the section (de Takats and Evoy Lymphedema Angiology, *1* 73, 1950)



FIG 241 FIG 242

FIG 241 A section of the skin and subcutaneous tissue in chronic lymphedema Note the enlarged lymph spaces which are valveless. Lymph circulates in them directed by gravity

FIG 242. Note that in addition to the enlarged lymph channels, the arterioles are thickened and the veins are collapsed as a result of increased tissue pressure

phosarcoma or Hodgkin s disease should be eliminated by a biopsy of palpable lymph glands and a roentgen film of the spine and pelvic girdle Unilateral lymphedema due to a hidden malignancy in the prostate uterus bladder or other visceral organs occurs frequently enough to warrant consideration On the other hand, not every lymphedema present in cases of obvious malignancy is due to a malignant invasion of the lymph glands A postphlebitic lymphedema frequently follows removal of malignant tumors and is primarily thrombotic in origin with an inflammatory component Radiation injury may seal lymphatics and produce lymphedema

Atypical, Mixed Forms of Lymphedema

POSTPHLEBITIC LYMPHEDEMA As described in the chapter on venous insufficiency a venous thrombosis especially of the iliofemoral segment will elicit a periphlebitic reaction—indeed an acute lymphadenitis—and the resulting edema is of mixed venous and lymphatic origin. As a matter of fact venous collateral circulation improves with time and the residual postphlebitic edema, especially the large patches of induration around the inflamed superficial and perforator veins, is due to periphlebitic inflammatory exudates which plug up the lymphatics and produce lymph stasis While the recent studies of Moyer and Butcher[21] emphasize the obliteration of dermal lymphatics in the presence of stasis ulcers our experience with wide



FIG 243 Subcutaneous tissue from Mary C , who suffered from an inflammatory lymphedema The elastic tissue is fragmented and there is irreversible fibrosis Nothing but excision can remedy such a situation

excisions of skin, indurated fat and thickened fascia indicates that all suprafascial lymphatics are clogged, that the fat is necrotic and that lime and bone deposits add to these postphlebitic indurations We have seen histologic pictures here identical with those obtained in inflammatory lymphedema (fig 244)

BARTENDER'S LEGS The combination of prolonged standing, chronic excessive consumption of alcohol with a poor nutritional state, hypoproteinemia and increased capillary fragility, produces swollen, almost intractable lesions to which skin infections, ulceration, self medication with antibiotics and lymphadenitis add a lymphatic component. Such men stand behind the bar for 12 to 18 hours daily and can hardly be rehabilitated without change in occupation

"ALLERGIC" LYMPHEDEMA With Dr J M Jesser, an allergist trained in vascular disease, we gave a considerable number of patients intracutaneous tests against fungi, streptococci and autogenous vaccines, but neither the diagnostic nor the therapeutic attempts have yielded any practical results, except that in patients who have repeated attacks of chills and fever a positive blood culture may be occasionally obtained An autogenous vaccine prepared from such a source seems to protect these patients from recurrent infections. Unquestionably, any sensitization phenomena, such as a sulfa-dermatitis or a penicillin-urticaria, are more readily localized in the lymphedematous extremity Allergens concentrate more readily in areas of poor lymphatic drainage

Lymph glands are at least one of the sites of antibody formation, since

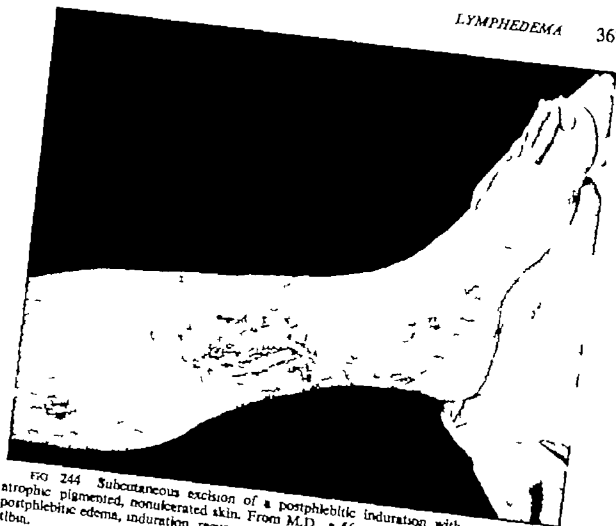


FIG 244 Subcutaneous excision of a postphlebitic induration with preservation of atrophic pigmented, nonulcerated skin. From M.D., a 56 year old man, who showed large postphlebitic edema, induration, recurrent ulceration, periostitis and sclerosing osteitis of the tibia.

they release lymphocytes containing gamma-globulin [22] Factors which elicit a release of lymphocytes from regional lymph glands are ACTH small doses of roentgen ray— and triple typhoid vaccine Triple typhoid vaccine may raise the lymphocyte count in an efferent lymphatic from 15 000 to 60 000 per cubic millimeter together with an enlargement and secondary regression of lymphoid tissue [23] Under the heading of therapy (p 372) this immunologic approach to certain types of lymphedema will be briefly discussed.

METHODS OF STUDY

Everyone who is confronted with the diagnosis and management of lymphedema has been looking for simple clinical measures whereby they can establish answers to the following questions (1) how much of the edema is fixed and due to irreversible fibrosis (2) is there a demonstrable segmental occlusion which could be removed or sidetracked and (3) can one visualize the enlarged lymphatics and the direction of flow in them

The amount of mobile fluid can be readily determined by steep elevation which may have to be maintained for a week and reinforced with a mercurial diuretic or Diuril (fig 245) As a matter of fact, this procedure must be

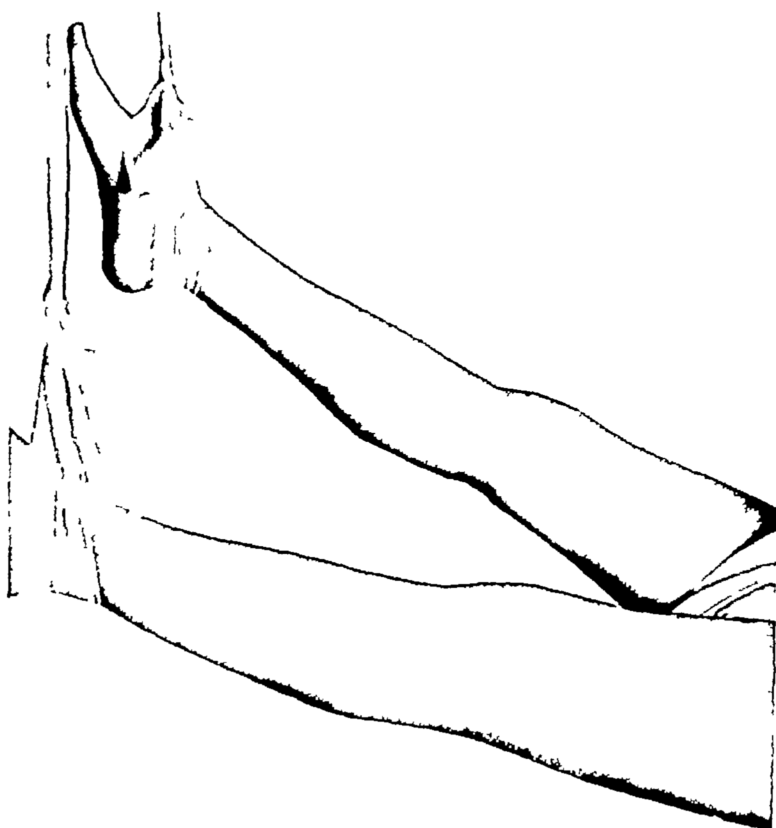


FIG 245. Elevation of a lymphedematous right lower extremity on a sling hung over a Balkan frame at an angle of 30° The left extremity was slightly elevated

utilized before any type of management, such as elastic compression or surgical excision, is undertaken

The level of segmental occlusion is often obvious, such as in a postmastectomy lymphedema or after a radical dissection of inguinal lymph glands, but it is often aggravated by secondary peripheral lymphangitis, retrograde spread of lymphatic thrombosis or metastasis and superimposed obstructive flare-ups of inflammation Opaque substances such as 35 per cent Diodrast injected into an enlarged tissue space occasionally may visualize a pool of lymph (fig 246) Thorotrast may be injected into a cannulated lymph channel at the ankle, but the opportunity to do this is slight and Thorotrast in our opinion should never be injected because of its prolonged radioactive property Colloidal iodine with procaine has been used, but is not a conclusive procedure The intradermal or subcutaneous injection of colloidal dyes started with the stimulating experiments of McMaster and his associates [10] They developed "streamers" in the skin and could demonstrate the rate of spread of the dye in different positions and on exercise We used this method in our Vascular Clinic some years ago, but reached no conclusions as to its practical value Recently, however, Moyer and Butcher[21] applied patent V. 5 dye (Sky blue, pontamine blue) to the study of postphlebitic ulcers and used it to demonstrate a lack of lymphatic circulation in the skin surrounding stasis ulcers

Histamine flares have been produced in the lymphedematous skin

around postphlebitic ulcers and were found to be absent perhaps because of a refractory state [24]

Flat soft tissue roentgen rays will demonstrate the coarse trabeculation of the soft tissues which never extends below the deep fascia Also four hours before surgery as outlined by Kinmonth and Taylor [25] 1 to 2 cc of sky blue are injected into the sole of the foot and the dye is then visualized on dissecting the lymphatics or the inguinal glands This has led to a frequent visualization of perivenous lymphatics but has not influenced our methods of procedure

More recently however Kinmonth and his co-workers[1] extended this visual lymphangiography to the injection of radio-opaque material into the lymphatic trunks previously stained with patent blue As a result of 87 lymphangiograms, 14 per cent aplastic and 55 per cent hypoplastic lymph trunks were found by these authors They have interpreted these findings to mean that congenital and juvenile types of lymphedema simply differ in the degree of hypoplasia of lymphatics and that injuries infections and pregnancy simply act to overload and decompensate the defective lymphatic circulation This is a highly important method of study of lymphedemas we have just started to use it



FIG 246 35 per cent Diodrast was injected into a large tissue space on the lateral aspect of the right thigh, which kept refilling with hemorrhagic lymph following a Kondoleon operation. A large lymph channel must have been draining into this tissue space which was finally obliterated by a sclerosing solution of Sotradecol.

TREATMENT OF LYMPHEDEMA

The objectives of treatment can be summarized as follows (1) mobilizing the edema fluid, (2) preventing the reaccumulation of fluid, (3) draining the lymph by sidetracking segmental obstruction, and (4) removing irreversibly damaged fibrous tissue with nondraining pools of stagnant lymph The success or failure of these measures will depend on the stage of the disease in which treatment can begin, whether the process of lymphatic obstruction is segmental or diffuse and whether it is progressive or recurrent

Treatment of Acute Lymphedema

In the acute stage of lymphedema, be it traumatic, inflammatory or allergic in nature, the following measures are of value to accelerate lymph drainage

POSTURAL DRAINAGE The upper extremity is hung from a Balkan frame, with care being exerted not to constrict the wrist in hanging it through a sling With the help of sedatives or narcotics patients are able to tolerate such a position overnight, and we have repeatedly rigged up such a device in the patient's home. Some patients will have to be relieved of this position for an hour every three to four hours For the lower extremity, the foot of the bed is raised 10 to 12 inches with the help of chairs or shock blocks. Pillows under the knee or under the mattress produce angulation at the knee and groin, and veins and lymphatics are not drained adequately Rose depics a wedge mattress[26] for the rapid elimination of edema from a lower extremity, this position is quite efficient, but often creates a sciatic type of neuralgia because of traction on the nerve

FREQUENT ACTIVE MOVEMENTS These movements in the form of muscular contractions are urged, whether the limb is in a splint or wrapped in foments, only in an acute, febrile, spreading lymphangitis is complete rest enforced

ADEQUATE ELASTIC COMPRESSION OF THE EDEMATOUS PART This is accomplished with elastic bandages, elastic adhesive tape, glycerin gelatin boots or the type of semirigid compression with Gauztex, which Norman E. Freeman[27] advocates A detailed description of these elastic or semirigid supports has been given in chapter 13 (p 342), since they afford ambulation Adequate, laced, leather sleeves are available on the market

DIURETICS In 1933, I described the rapid disappearance of thrombophlebitic edema with the help of mercurial diuresis [28] In the light of our present knowledge, the retained tissue fluid is mostly due to lymphatic blockade Acute lymphedema will respond after a few days of acidification with ammonium chloride or nitrate to one or two injections of Mercuhydrin Recently, in a few patients, Diamox has been used in daily 250 mg doses, especially as a rapid preparation for surgical procedures in the lymphedematous limbs It is important not to dehydrate a patient to the extent that his clotting mechanism will be disturbed As pointed out in chapter 12,

Thromboembolism excessive diuresis in cardiac patients may bring on arterial and venous thromboses Diuril can also be used in 500 mg doses twice daily

Restriction of sodium chloride to 1 to 2 Gm daily without restriction of fluid intake is a reasonable measure In the subacute and chronic types of edema an excessive intake of beer salty appetizers or mixed alcoholic drinks noticeably increases the swelling

SYMPATHETIC BLOCK. In acute lymphedema there is no evidence of vasospasm The pulses are bounding and the skin is warm often flushed Oscillations are increased and the plethysmograph shows increased pulsations Therefore help cannot be expected from paravertebral blocks unless evidence of vasospasm is present or unless the edema is part of a causalgic state (chapter 16) where pain relief in the early cases is dramatic

HEPARIN THERAPY Zimmerman and I[3] noted a massive fibrinous exudate in the muscles and subcutaneous tissue and found a high protein content in the tissue fluid when acute thrombophlebitic edema was produced Heparin acts on this fibrinous exudate and it would be difficult to explain the sudden decrease in acute thrombophlebitic edema following the administration of heparin unless it acted on the lymphatic component of thrombophlebitic edema The late John Homans always insisted that the mechanism of the large white edema in major venous occlusions is due to lymphatic blockade and although our initial experiments with Zimmerman seemed to implicate a pure venous obstruction the wisdom and accuracy of Homans early teachings are now fully obvious [29] Heparin then as pointed out by Loewe and his associates [30] may well act on this extravasated plasma and facilitate its uptake into the lymphatics If administered early enough it should militate against thrombosis in the lymphatics and against the fibrinous exudate in the sinusoids of the lymph glands

For these reasons a priming intravenous dose of heparin is given amounting to 50 to 100 mg. Within an hour capillary coagulation times will be raised to between 8 to 12 minutes This elevation can now be maintained by intramuscular injections of 100 mg. of heparin in 10 per cent aqueous solution (1 cc) every 8 to 12 hours Four to five days of this regime should suffice provided the patient can be mobilized Should he have to be bed ridden for a longer time this type of heparin administration can be maintained with daily doses of 100 to 200 mg of heparin depending on the patient s individual response

Heparin therapy for acute lymphedema is obviously contraindicated in traumatic cases where hemorrhage would be increased or in the rapidly spreading tubular type of lymphangitis where it would interfere with the protective effect of fibrinous fixation [8] We have been impressed with the rapid decrease in the edema of inflammatory exudates in an acute gouty arthritis and even in an acute periarthritis with synovitis. This anti inflammatory antiphlogistic effect of heparin will be discussed later it may not be purely dependent on its anticoagulant effect Fibrinolysin given intravenously

in a recent case produced a dramatic disappearance of an acute postphlebitic lymphedema

ROENTGEN RAY THERAPY In 1933, Zimmerman, Gault, Halpern and I[31] described the rapid disappearance of experimentally produced thrombophlebitic edema following radiation More recently we have reported on 100 patients suffering from acute lymphatic or periphlebitic obstruction, who received one to several exposures to roentgen ray [32] Roentgen ray hastens the absorption of inflammatory exudates and produces destruction of lymphocytes While large doses have a direct effect on the lymphocyte, these should be obviously avoided, the general rule that the more acute the inflammation, the smaller should be the dose, must be strictly observed Severe febrile reactions can occur when 150 to 200 r are being given, from 50 to 80 r seems to be the optimal dose for most acute and subacute cases of lymphangitis and lymphadenitis It has been our custom to observe the effect of a single dose for several days to one week, and then administer the next dose One to three treatments usually suffice for each area, using 20 kv , 20 ma , fsd 50 cm. and a medium Thoreus filter The hvl is 1 9 mm copper.[32]

TRIPLE TYPHOID VACCINE The effect of nonspecific protein therapy on the course of infections by injections of milk, autohemotherapy and other parenteral proteins, has long been known It has been thought that the patient's natural resources of resistance were thereby enhanced It is also known that triple typhoid vaccine, whose dose can be more easily graded than any other such material, raises the number of lymphocytes in the lymph channels draining a lymph node and that gamma-globulin in the blood is thereby increased [23] In patients suffering from an acute inflammatory hyperplasia of the lymph glands, the sinusoids can be cleared by such parenteral protein therapy, and with small doses of triple typhoid vaccine, containing not more than 100,000 to 500,000 bacteria, one can "wash out" the lymph glands and prevent permanent blockade It is likely that the vaccine acts through stimulating the pituitary-corticoadrenal axis

Treatment of Chronic Lymphedema

When one looks at the histologic sections of tissues afflicted with chronic lymphedema and sees the heavy fibrotic strands in the subcutaneous fat, the enlarged tissue spaces in which the fluid undulates to and fro with gravity, the thickened deep fascia which barricades the lymph-soaked tissue from the muscle spaces and the blocked, fibrinous sinusoids of the lymph nodes which are impermeable, it becomes clear that the process is irreversible and that the object of treatment can be either an arrest of the disease to check further deterioration or the removal of as much of this tissue as possible without sacrificing the function of the limb In the exceptional case, a segmental block in lymph drainage may be bypassed, and in some extreme cases amputation may become necessary

It has been our custom to hospitalize such patients for a few days, using maximal elevation of arms or legs together with mercurial diuretics or Diamox In a few days it will then become obvious how much fluid can be

mobilized and how much of the enlargement is fixed A well fitting elastic hose several elastic bandages or a semirigid Gauztex bandage can now be applied and the patient is ambulated while still in the hospital to see how well the swelling can be contained and whether a looser elastic support will be sufficient Salt is restricted to 1 to 2 Gm a day and massage at home by members of the family—instructed and occasionally supervised by a physical therapist—is prescribed All sources of infection from fungi in the feet, from a vaginal source or from other foci are treated Anemia hypometabolism and hypoproteinemia are corrected if possible With such a regime chronic lymphedema may often be kept in check and while in women it does constitute a considerable handicap and source of emotional strain many patients can be kept comfortable and economically independent thus invalidism and mental fixation on a cosmetic problem are prevented Many of our women patients with congenital and inflammatory lymphedema have married borne children and are well adjusted to this deformity

A serious complication develops however when intermittent attacks of chills, fever and an acute flare up of an erysipelas like skin reaction supervene The attacks may occur once every few years but then as often as once every two or three months sometimes they follow a mild respiratory reaction or a mild trauma but most of the time they are without an obvious cause At the time of the chill or shortly thereafter a positive blood culture may be obtained we have only obtained this three times, although blood was cultured in many instances When an autogenous vaccine is thus available 0 1 to 0 2 cc of such a vaccine are given every four to five days for a series of twelve injections Whether this method is really a safeguard against future flare ups is uncertain but we have not seen any flare ups in the vaccinated cases

When an autogenous vaccine is not available a polyvalent streptococcus vaccine has been advocated However we have not availed ourselves of this method especially since the offending organism does not have to be a streptococcus Rather we have placed such patients on long term antibiotic therapy notably Gantrisin starting with daily 3 to 4 Gm doses for a week and then continuing with 1 5 Gm doses daily for many months suspending the treatment on Saturdays and Sundays

In many younger women the appearance of a large disfiguring unilateral edema of the leg, which may gradually spread over to the opposite side is most discouraging in fact it may lead to a state of mind which makes their lives miserable This situation together with the continued increase in size and weight and the imminent breaking down of the skin with hyperkeratotic patches, are indications for a surgical procedure We have not advised surgery for the milder cases for those in whom good elastic compression keeps the edema under control or for those who expect a cosmetic result since this rarely ever happens Surgical procedures are only undertaken when the lymphedema is obviously getting out of hand when it produces disability or when there is danger of losing the limb which does infrequently occur

The surgical treatment of lymphedema consists enssetially in removing the fibrotic, lymphedematous, inflamed areas from the lower leg and foot, and less frequently from the thigh The idea of draining lymph to the subfascial muscle spaces, where it can be more readily absorbed, originated with Handley, who placed silk threads in the subcutaneous tissue [33] In our clinic, W. C Beck experimented with the placement of Nylon threads into arms which were lymphedematous after mastectomy, and modest results have been reported by Guthrie with this method [34] We have not pursued this method any further and no other reports are available Another approach was that of Kondoleon,[35] who produced a small window in the deep fascia, removed some of the edematous subcutaneous tissue and fat and replaced the skin on the muscle

Our initial attempts followed the methods of Sistrunk[36] and Homans,[37] who proceeded to enlarge the fascial windows, remove more and more lymph-soaked tissue and finally remove the lymphedematous skin (Macey[38]) These procedures are all based on the premise that the lymphedema is suprafascial and that there is no lymphedema in the subfascial muscle compartments Evoy and I[11] reported on the results of an operation, essentially that of John Homans, which has had moderate success in keeping down the edema, although we well realize that the control of the blockade of lymph has not been relieved and that much reliance needs to be placed postoperatively on elastic support.

As will be described in chapter 21 (p 598), there are basically two surgical objectives in treating lymphedema. One of these is to bypass or overcome an obstruction to the flow of lymph, if this is localized to a segmental area Thus, pressure or obstructive lymphangitis in the region of the cisterna chyli has been relieved by implanting a large lymphatic duct into the hemiazygos vein [39] This seems like an ingenious procedure, however, although we have systemically searched for central obstructions, we have only occasionally encountered lymph cysts or a chylous reflux from the intestine with a lymphedematous bowel and we have never really been able to remove a central block. In the lymphedemas caused by malignant metastatic spread to the inguinal, pelvic or retroperitoneal nodes, appropriate doses of deep roentgen ray therapy may relieve pain and some edema

Gillies and Fraser[40a] grafted a lymphatic-bearing bridge of skin from an arm to a leg or from one arm to another Sir Harold Gillies presented a photograph of a patient 15 years later, showing an excellent result from this "lymphatic wick "[40b] However, the majority of surgeons, including plastic surgeons, have not followed this lead Because of Kinmonth's latest work,[1] obstructive lymphedema is often not the primary cause of lymphedema

In the overwhelming majority of patients, however, the difficulty is due either to an ascending obliterative lymphangitis or to a lymphatic defect or valvular insufficiency due to postural lymphatic reflux in the erect position Here excision of the lymph-soaked area is the goal In the case of Dolores W , a 16 year old girl with a congenital lymphedema and very little fibrosis,

an excellent result was obtained The picture was taken one year after sur gery No elastic support has been necessary (fig 247)

Our standard procedure has been the Kondoleon operation as extended by Servelle [41] who employs a total resection of the deep fascia from foot to knee and maintains the skin flaps The medial aspect of the right and the lateral aspect of the left lower leg are operated on either simultaneously or one week apart The stitches have just been removed two weeks after the operation in figure 248 Glycerin gelatin casts are applied once a month for three or four months before operations are continued on the contralateral sides The thighs are seldom subjected to these wide excisions Curiously enough, in the last two instances in which long excisions have been made on the lateral aspect of the thigh long lasting exudates of hemorrhagic lymph have resulted necessitating aspiration of 60 to 100 cc of lymph from under the skin It was finally necessary to clot the lymph in one instance with the patient s own blood and in another with a sclerosing solution of 5 per cent Sotradecol Such an occurrence demonstrates of course that the basic obstruction or insufficiency of the lymphatic pathways has not been relieved

In the postphlebitic lymphedemas, in which the induration and ulcera tion are actually due to obliteration of the dermal lymphatics [1] extensive removal of the epithelium at a 15/1000 to 16/1000 of an inch depth seems important. But in the typical congenital lymphedema we have preferred to preserve the skin flaps While Poth Barnes and Ross[42] and Pratt[43] have emphasized the removal of the skin of lymphedematous legs, we have reserved this procedure for patients in whom the skin itself was, to the naked



FIG 247 Congenital lymphedema In Dolores W one year after surgery The right lower extremity contained a number of large lymph cysts. No elastic support has been nec essary (de Takats and Evoy Lymphedema. Angiology *1* 73 1950)



FIG 248 Kondoleon operation in Mrs Elizabeth E The stitches have just been removed The epidermal crusting at the left ankle readily peels off and does not lead to deep necrosis The ankle is a frequent site of marginal necrosis (de Takats and Evoy Lymphedema Angiology, *1* 73, 1950)

eye, visibly indurated, thin, atrophic and vulnerable Tests with histamine or patent V blue may be employed (p 368), but they generally coincide with what is seen on simple inspection and palpation

For lymphedematous legs, however, whose skin is excoriated, eczematous, ulcerated or obviously vulnerable, the removal of all skin with an electric dermatome is followed the removal "en masse" of the subcutaneous tissue, fat and fascia (described by Pratt[43])

Our results in 28 patients followed from 1 to 25 years were reported in 1950[12] (Table XI) Since that time, and with the use of the electric derma-

Table XI

RESULTS OF SURGICAL TREATMENT OF LYMPHEDEMA IN 28 PATIENTS FOLLOWED FROM 1 TO 25 YEARS

NUMBER OF PATIENTS	RESULTS
17	Satisfactory
5	Doubtful
6	Poor

Satisfactory disease is arrested, the limb has excellent function and there is no economic handicap *Doubtful* there is some return of edema, recurrent inflammation and a reoperation is contemplated or is in progress *Poor* there is progress of the disease, ulcerations and the limb is a severe handicap to the patient

tome, our end results are somewhat better It is obvious however that early surgical attack and a better understanding of the impairment in lymphatic circulation may vastly improve the results Generally speaking, the prevention of an acute pitting edema from developing into a hard fibrous irreversible one is far more rewarding than the treatment of the full blown forms of edema

Occasionally amputation is inevitable especially in patients who show involvement of the pelvis and genitalia (fig 249)

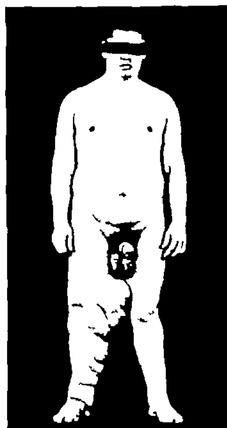


FIG 249 D L is shown at age 21 after multiple Kondoleon operations had failed. A high thigh amputation followed by intensive treatment in the Department of Physical Therapy and Rehabilitation enabled him to work as an accountant.

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CHAPTER 15

ANEURYSMS

REFERENCE HAS ALREADY BEEN MADE IN THE FIRST PART OF THIS MONOGRAPH to the ingenious studies of Emile Holman,[1] who showed that "structural fatigue" develops in arterial segments distal to a narrowed portion and that the poststenotic dilatation seen in aneurysms distal to subaortic stenosis, pulmonary valvular stenosis, coarctation of the aorta or beyond a cervical rib can be produced in a model experiment in which fluid is pumped through a rubber tube for a sufficient length of time The turbulence of the blood and the transformation of high kinetic energy to lateral pressure exert a continuous pressure on the wall of the vessel

There are, however, other initiating or accelerating factors, since not all aneurysms show a demonstrable proximal stenosis These have been enumerated in a previous communication by Pirani and myself[2] The first response to *increased intraluminal pressure* is elongation, as long as the arterial segment retains its full elasticity, it returns to its original shape when the tensile force is withdrawn When the limit of elastic recoil is exceeded, a permanent deformation will develop in the form of elongation and dilatation. In aortograms of hypertensive patients, one frequently sees elongation and left-sided convexity of the abdominal aorta fixed between the diaphragm and the iliac bifurcation (fig. 250)

The smaller the artery, the higher the pressure it tolerates In the dog, at least, the aortic arch ruptures at twice the atmospheric pressure, the descending aorta will tolerate four times and the carotid artery seven to eleven times the atmospheric pressure[3] The diameter of the vessel wall has a marked influence on the tensile load acting on its wall The Law of Laplace (1841), discussed and illustrated in the first part of this monograph (p 5), indicates why the dilatation of an arterial segment increases the lateral pressure exerted on its wall Thus, every aneurysm contains in itself a tendency to grow continuously until it ruptures This has a direct bearing on the indication to operate, whenever the patient's general condition permits

During a sudden rise in blood pressure, a normal vessel first expands and then diminishes in size These changes are due to the behavior of the muscular and elastic elements A rigid, fibrosed, inelastic wall will not do this and hence will continue to yield to intraluminal thrusts of systole The loss of elasticity will lead to an increase in pulse pressure and an increased volume of blood in the aorta While the velocity of the pulse wave is con-

siderably more rapid than that of the blood, the velocity will increase as age increases [4]

All these observations lead us to the focal point of elasticity By elasticity of an artery is meant the percentual increase in the volume of the artery with each millimeter increase in blood pressure It is related to the velocity of the pulse wave thus

$$\text{elasticity} = \frac{1\ 72}{\text{velocity of pulse wave}}$$

Since there is a notable decrease in elasticity as age advances and since less fluid can be accommodated by distention of the arterial system the same cardiac output will raise the blood pressure during systole to higher levels in older than in younger individuals [5] Loss of elasticity is also to be expected in homologous arterial grafts [5b] However this has nothing to do with tensile



FIG 250. Aortogram in a 52 year old hypertensive patient. Note the elongation and tortuosity of the abdominal aorta and the iliac vessels. Questionable stenotic areas are in the midportion of both renal arteries. (de Takats, G and Pirani, C. L. Aneurysms General Considerations. Angiology 5 173 1954)

strength, since enzymatically digested bovine arteries are nothing but collagen tubes with no elastica and yet they show great tensile strength and are flexible [5c]

In addition to these general principles, local causes responsible for segmental dilatation and saccularity are in operation Leriche[6] postulated a neurogenic basis for large dilated iliac or femoral arterial segments which showed no obvious obstruction above or below the lesion Experience with aneurysms developing distal to cervical ribs dates back many years and has attracted the interest of Halsted [7] While occlusion of the vasa vasorum will be discussed in the next chapter, a hydrodynamic principle is also at play. A nozzle effect was emphasized by Blakemore, in his studies on partial constriction proximal to abdominal aneurysms,[8] he pointed out that unless the lumen is constricted to 75 per cent or more of its original diameter, more harm than good can come from partial ligation On the other hand, when the lumen of the aorta is reduced by more than 55 to 60 per cent, an elevation of aortic and left ventricular pressures can be produced in the experimental animal [9] Thus, it must be remembered that by treating aortic aneurysms by partial proximal constriction, the hemodynamic effects of coarctation may be produced, even though the acute experiment does not simulate a chronic subrenal aortic stenosis as produced by partial ligation of an abdominal aneurysm This indicates the highly delicate adjustments to be made when partial constriction is planned The method, however, has been abandoned

In coarctations of the aorta, Robert Gross encountered 21 aneurysms in 270 patients who have come to operation [10] Of these 21, about half of the subjects had an aneurysmal dilatation of the aorta below the block, the aneurysms varying in size from a small hen's egg to a large lemon In the remaining patients, the aneurysm was in the intercostal artery just as it joined the aortic wall

The pathogenesis of the aneurysm distal to the coarctation is believed to be a jet effect exerted through the narrow lumen, but the lumen must be at an angle with the long axis of the aorta The bulbous dilatations at the entrance of the upper intercostal vessels and the traction of the ligamentum arteriosum, together with back pressure from below, are the factors enumerated by Abbott [11]

If one were to clinically apply any of the simple hemodynamic principles just enumerated, the following points will need to be stressed

(1) Hypertension always complicates the treatment of aneurysms because of the increased strain on the aneurysmal wall A decrease in blood pressure by medical or surgical means is desirable

(2) A decrease in the diameter of the sac by intraluminal clotting, by extraluminal support or by partial excision decreases lateral tension on the wall

(3) Proximal partial constriction has to be intense (at least 75 per cent), otherwise it causes more of a spraying effect and does more harm than good

(4) Removal of the entire dilated and diseased segment and its replacement by a homologous arterial graft or a plastic prosthesis restores normal

hemodynamic conditions, unless the vascular disease is too diffuse to cope with

STRUCTURAL DEFECTS LEADING TO ANEURYSMS

Although all of the elements which constitute the arterial wall are important for the normal function of the arteries, emphasis will be placed in this discussion on those structures which appear to play a more significant role as far as resiliency and nutrition of the vessel wall are concerned and which seem to be more directly involved in the pathogenesis of aneurysms

The wall of a normal artery in a young adult starting from the lumen consists of a single layer of endothelial cells, a thin layer of collagen fibers which together with the endothelium forms the intima a thicker layer consisting of smooth muscle fibers and elastic fibers forming the media, and an outer layer or adventitia consisting of relatively loose connective tissue in which are scattered elastic fibers and which contains blood vessels and nerves

A *normal intimal endothelium* is essential for an undisturbed flow of blood through the lumen of the vessel. Any irregularity of the endothelial surface may lead to deposition of fibrin platelets or other hematogenous elements This process may be extremely important in the pathogenesis of arteriosclerosis and atherosclerosis as recently stressed by various investigators [12, 13] Endothelial cells are large flat elements in close apposition one to another, although possibly separated by a cement substance (fig. 251) The existence of this material presumably a mucopolysaccharide has recently been convincingly demonstrated [14] Endothelial permeability either by active cellular function or on the basis of simple passive diffusion is, of course extremely important for the nutrition of the vessel wall Precise information on this point however is extremely meager

The subendothelial connective tissue or the intima proper is a loose framework of collagen and elastic fibers separated by ground substance with a few scattered fibroblasts It is capable of great proliferation and of accumulating within itself many different materials of hematogenous origin The latter property appears to be dependent to a considerable extent on the state of the endothelial lining The role of the ground substance in the intima and in the media is of considerable importance and will be fully discussed later

The tunica media consists of elastic and smooth muscle fibers in different proportions depending on the size of the artery Ground substance fills the interstices between the fibrillar elements As a rule, collagen fibers are absent, and fibroblasts are only occasionally seen in the media of normal arteries It is generally agreed that muscle fibers, while providing for the contractile properties of muscular arteries and for the maintenance of vascular "tone contribute relatively little to the over all tensile strength of the arterial wall This appears to be based predominantly on the collagen fibers and on the connective tissue of the adventitia Elastic fibers are arranged in fenestrated concentric lamellae which, at their point of maximal concentra

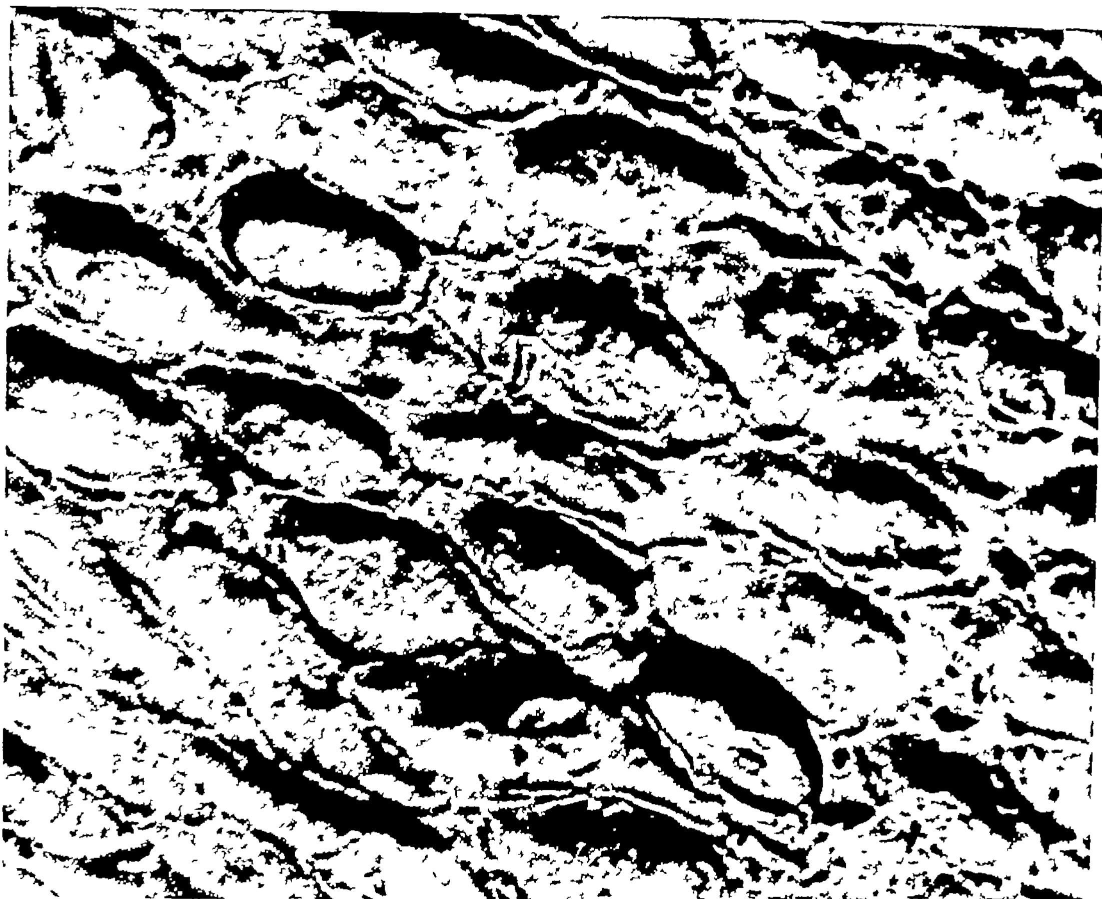


FIG 251 Stereoscopic view of normal endothelium The lines correspond to the intercellular cement (Altschul, R Endothelium The Macmillan Co , New York, 1954)

tion, form the internal and external elastic membranes In the aorta there are approximately 50 to 60 fenestrated lamellae in the media, measuring on the average 2 5 μ deep In the smaller arteries, elastic fibers are considerably more delicate and less numerous, while the elastic membranes are better developed and defined [15] Elastic fibers must be considered a highly specialized product of interstitial mesenchymal tissue They consist of elastin, which is fundamentally a peptide chain with characteristic histologic, chemical and physical properties [16] There is little doubt that elastic fibers, with their considerable tensile strength and rubber-like elastic properties, provide much of the resiliency of the arterial wall The arrangement of the elastic laminae is such that it provides an equal distribution of mechanical loads on the entire surface and thickness of the vessel wall However, their fenestrations may represent potential points of weakness, especially if the ground substance filling the interstices undergoes changes, either in their quality or quantity It is well known that elasticity of the arteries and especially of the aorta decreases with age [17] There is no definite relationship, however, between loss of elasticity and incidence or severity of arteriosclerosis Severe arteriosclerotic and atherosclerotic lesions may be found in arteries which have almost completely maintained their elasticity, and conversely no arteriosclerotic lesions may be found in aged individuals whose vessels have lost almost all their elasticity Loss of elasticity which is accompanied by diminution of ten-

sile strength does not appear to be related to the quantity of elastic tissue in each unit volume of the aortic wall, since this remains nearly constant throughout life [18] Local atrophy and disintegration of elastic lamellae but especially the increased amount of ground substance in the arterial wall are probably responsible for the diminished elasticity of the aged aorta

The presence of ground substance in the wall of normal arteries, especially of the aorta, has been known for some time but its importance has been re-emphasized only in recent years This material consisting mainly of acid mucopolysaccharides is present in the intima and in the media appearing in greater concentrations near the internal and external elastic membranes, but also filling, at least in part the fenestrations of the elastic lamellae This distribution is particularly obvious in tangential sections of the aorta It is generally accepted that the mucopolysaccharide of the aorta has the same physical characteristics of mucoid substances elsewhere in the body Its origin has not been definitely established although it appears likely that the fibroblasts are responsible for its formation [19, 20] The widespread distribution of ground substance in the arterial wall has suggested that this material plays an important part in the nourishment of the embedded elements particularly of the elastic fibers with which it is in close association This possibility is strengthened by the fact that metachromatic substances are found chiefly in poorly vascularized tissues, not only tissues in arteries but also those throughout the body According to some workers this material is an effective cement substance from which collagen and elastic fibers are maintained and formed In addition because of its physical character it has been suggested that the mucopolysaccharide permits a frictionless rhythmic shifting of the elements of the vessel wall which are embedded in it This view appears likely since ground substance is more heavily concentrated in the inner part of the vessel wall, which is probably the area of greatest mechanical shift during expansion and contraction due to the pulse wave It is interesting to note that the mucopolysaccharide increases in the media with age reaching a maximum in the third or fourth decade Its amount then remains fairly constant for two or three decades, then declines progressively in the older age group together with atrophy of the various elements of the vessel wall and the loss of elasticity It is also well known that ground substance is increased in amount in arteriosclerotic and atherosclerotic areas, as well as in the media in some cases of dissecting aneurysm It is generally conceded that accumulations of ground substance in the intima and media are actually the result, and not the cause of degenerative changes taking place in the two layers of the vessel wall Nevertheless it is likely that large local accumulations of ground substance associated with gaps in the muscular coat and interruption of the elastic fiber network represent a locus minoris resistentiae" and may very well be the initial site of aneurysm formation at least in some cases

The outer layer of the arteries or the adventitia has characteristics of considerable interest in relation to the problem under consideration The adventitia although basically a loose structure containing a relatively small

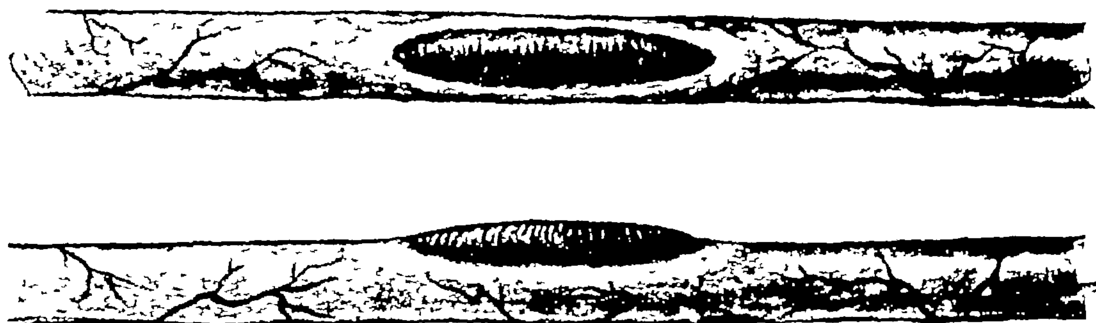


FIG 252 Bulging of the arterial wall after stripping of the adventitia (Winternitz, et al [17])

number of elastic and collagenous fibers, is provided with great tensile strength It has been shown that removal of the adventitia of the carotid artery of dogs in vivo resulted either in rupture or in a fusiform dilatation of the vessel[17] (fig 252) Under this condition the unrestrained medial coat bulges through the adventitial gap with each systolic impact It would appear, therefore, that the tunica media alone, in spite of its well developed elastic and contractile properties, is not adequate to cope with the systolic blood pressure and that the tunica adventitia is essential for the maintenance of a normal arterial caliber It should be emphasized that the adventitia is a vascular tissue containing arterial and venous vasa vasorum derived from either the main vessel or more commonly from secondary branches This vascular network is extremely well developed, especially in larger arteries, and may play a role in the establishment of collateral channels when the lumen of the vessel is narrowed or occluded The predominant origin of vasa vasorum from secondary branches must also be kept in mind, since ligation or obstruction of these trunks might presumably lead to a circulatory disturbance, relative ischemia and damage to segments of the main vessel below the ligature A similar effect might be obtained by infiltrative or destructive processes extending to the adventitia from adjacent areas Experimental proof of this possibility has been provided by the studies of Schlichter[21] who, by cauterization of the adventitia, was able to produce medial necrosis with resulting aneurysmal dilatation and arteriosclerotic lesions in the corresponding segment of the arterial wall

The vasa vasorum have, therefore, an important function in the nutrition of the arterial wall, and then appear to play an important role in the pathogenesis of arteriosclerotic lesions and syphilitic arteritis as well It has been shown by a number of investigators[17, 22] that, in normal arteries, vasa vasorum of adventitial origin penetrate as far as the inner third of the media, while the intima is an avascular structure Although the lack of demonstrable capillaries in the intima is not absolute, it is generally agreed that this portion of the arterial wall receives its nutriment directly from the main lumen by permeation of plasma constituents through the endothelium This physio-

logic mechanism is apparently regulated by factors of endothelial permeability filtration pressure and probably by the status of the ground substance of the intima On the other hand circulation through the vasa vasorum is dependent on the blood pressure in the particular artery under consideration and on the state of contraction or tonicity of the blood vessel It appears likely that blood circulation through the vasa vasorum is not continuous being comparable in this respect to that of the myocardium through the coronary arteries According to this concept, active circulation in the vasa vasorum would take place during diastole and very little or no blood would reach the media when the arterial wall is stretched by the systolic impact Changes in heart rate particularly tachycardia, in blood pressure and in tonicity of the vessel wall which is regulated by autonomic nervous impulses probably interfere with a normal circulation through the vasa vasorum

With arterial aging and the development of arteriosclerotic lesions, important changes occur in the distribution of vasa vasorum in the arterial wall A moderate intimal thickening is known to occur with age mainly as a result of the continuous blood pressure impact and of deposition of ground substance A more severe thickening of the intima however is often caused by deposition of hematogenous substances such as fibrin on the endothelial lining.[12] Fibrin is either incorporated in the intima as such or will lead to formation of a mural thrombus which in the course of time will become organized and vascularized In later stages, fibrosis, hyalinization and endothelialization of the thrombus will make it impossible to separate histologically this lesion from an intramural vascular lesion due to other causes Whatever the cause of intimal thickening, if this is sufficiently marked the intima will acquire a network of capillaries originating either from the lumen or by extension of the adventitial plexus through the media and the internal elastic membrane [23] Often vascularization occurs on the basis of both processes and in this case anastomoses between the two networks of capillaries develop These newly formed capillaries and anastomoses at the medial intimal junction, because of their relatively large caliber and perhaps imperfect structure are more easily subject to secondary disturbances. Hemorrhages which are known to occur within the wall of arteriosclerotic arteries are more likely to occur at this site These hemorrhages are considered by some to be true infarcts and may be the result of vasa vasorum thrombosis, sudden lowering of arterial pressure or arterial spasm which would interfere with intramural circulation Thromboses within the main lumen may develop over the atherosclerotic plaque, and by obliterating the origin of the intimal vasa vasorum may initiate or aggravate the process In addition, the vasa vasorum, especially the arteriolar branches of the adventitia, may show thickening, fibrosis and hyalinization of their wall with narrowing of their lumen This process is observed not uncommonly in cases of severe hypertension with generalized arteriolar sclerosis and is often seen in association with large atherosclerotic plaques and aneurysms (fig. 253) *In syphilitic arteritis* endarteritis of the adventitial vasa vasorum with narrowing of their lumen is a prominent feature and will be referred to later

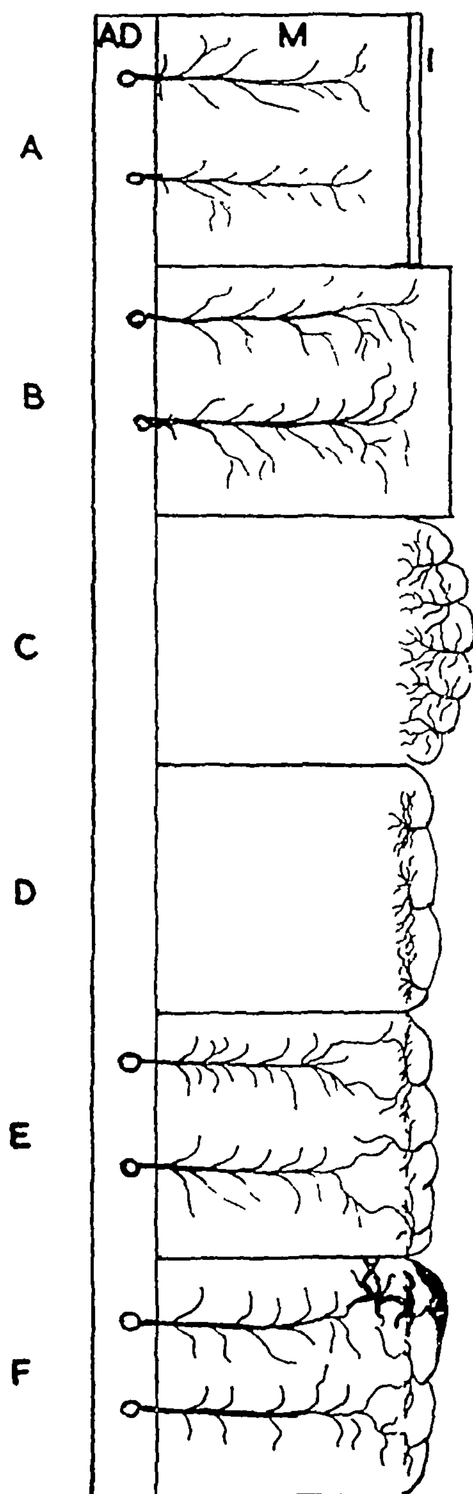


FIG 253 Arteriolar changes of the vasa vasorum in hypertension with arteriosclerosis (A) Vasa vasorum from the adventitia terminate normally at the middle third of the media (B) Transmedial vascularization occurs when the intima is thickened (C) Recent mural thrombus is on the intima The vascular channels communicate with the lumen (D) Degeneration and shrinkage of the mural thrombus Note the vascularization from the lumen, an "inverted tree" appearance (E) Combined vascularization occurs from the adventitia and lumen, with anastomoses of the two systems (F) There has been a vascular accident in the combined form of vascularization A microinfarct and deposition of fresh thrombus are on the intima (Modified from McLetchie, N G B The Pathogenesis of Atheroma Am J Path, *28* 413, 1952)

Anoxia of the arterial wall is, therefore, extremely important as a factor in the pathogenesis of arteriosclerosis, and it appears to play a role in weakening the arterial wall in this disease as well as in syphilitic arteritis To summarize vasoconstrictor influences, change in intravascular pressure, colloidal plasmatic disturbances resulting in the formation of fibrin and precipitates over the intima and hematic anoxemic agents are the factors involved in this process either alone or in combination [24] It may be that accumulation of lipids in the arterial wall is predominantly a secondary phe-

nomenon being the result of medial necrosis and hemorrhages physico-chemical alterations of the ground substance and increased endothelial per meability although disturbances in the lipid metabolism have recently been intensely studied

Having briefly reviewed the various elements which constitute the ar terial wall and pointed out some of their functional properties, we can now apply this knowledge in an attempt to clarify the pathogenesis of the various types of aneurysms

THE PATHOGENESIS OF ANEURYSMS

Arteriosclerotic aneurysms are at present the most common type and with the decreasing incidence of tertiary syphilis and the increased life ex pectancy of the population they can be expected to have an even higher incidence in the future [25] As just discussed various factors contribute to damage the arterial wall in arteriosclerosis The great emphasis placed on studies of lipid metabolism may not solve the over all problem of this dis ease although, of course it may clarify a number of important points It is our feeling, based on the reports of numerous investigators that the local factors such as deposition of fibrin and other hematogenous substances in the intima accumulation of ground substance disruption of elastic lamellae in the media, and especially disturbances of circulation in the vasa vasorum either on a functional or anatomic basis or both play the initial and perhaps predominant role in this disease Of course atheroma formation may further weaken the arterial wall and atheromatous ulcers in particular may in some cases be the basis of aneurysm formation Foci of fibrosis and scar ring of the arterial wall with or without lipid deposition will result in the formation of inelastic and noncontracting areas Similar to what happens to the heart in cases of myocardial infarct, these areas do not contribute to the work performance of the artery involved and tend to be stretched by me chanical forces dissipated against them [26] Arteriosclerosis and atherosclerosis are more frequent and more severe where arteries divide where new branches take off or where inflammatory adhesions or other processes fix the arterial wall to adjacent structures [27]

Once the initial dilatation of the arterial wall has taken place the course of the disease is progressive Formation of mural thrombi within the aneu rysmal cavity and extensive fibrosis of the arterial wall and of the periarterial tissues may delay expansion and rupture of the aneurysmal sac, but cannot prevent the inexorable progress of the lesion At this stage three factors appear to be involved in determining the progress of the disease the level of the systolic blood pressure the direction and force of the secondary blood currents within the aneurysmal sac and the state of vascularization of the aneurysmal wall The importance of the first factor is self evident that of the second can be explained by mentioning one of the laws governing the flow of liquids through tubes, *i e* lateral pressure is inversely proportional to the velocity of the liquid The third factor is the least easily understood

and least accessible to therapeutic procedure It is generally accepted, however, that the wall of an aneurysm is poorly vascularized and, as a result, in most cases the fibrous tissue replacing elastic and muscle fibers is defective or inadequate or both, and the mural thrombi at best undergo only partial organization. In resected aneurysmal sacs one frequently sees liquefaction or actual necrosis of the thrombus and of the thin wall it protects. This is the site of rupture. Artificial induction of a solid fibrous connective tissue capsule around the aneurysm has been tried in the past but is only justifiable now if no direct attack on the aneurysm is possible

In syphilitic aortitis diffuse dilatation of the aorta and the formation of saccular aneurysms are the most serious complications. The problem here is quite different from that of arteriosclerosis. In the first place, the etiology of the disease is known and, as a result, prophylactic and therapeutic measures against the etiological agent have already greatly reduced the incidence of the disease and its complications Second, aneurysm formation is generally agreed to be a much more common complication of syphilitic arteritis than it is of arteriosclerosis. However, the pathogenesis of syphilitic aneurysms, most commonly located in the ascending and transverse portions of the aortic arch, needs some clarification The development of syphilitic aneurysms is dependent essentially on two factors. The first is the active inflammatory destruction of all elements of the arterial wall at a rate which exceeds that at which the damage can be repaired by fibrous tissue proliferation [28] Destruction of elastic and muscle fibers of the media, either alone or in combination, as previously pointed out does not necessarily result in a serious loss of the total strength of the arterial wall It should again be stressed that unless all layers of the artery including the adventitia are involved, aneurysmal dilatation probably would not occur The second factor involves again the vasa vasorum which, in most cases, are the site of severe endarteritic lesions with consequent poor circulation and anoxia of the vessel wall As a result of the active inflammatory process and of poor nutrition, proliferation of connective tissue and aneurysmatic dilatation will follow In this process, blood pressure, especially if elevated, plays an important role The predominant location of syphilitic aneurysms in the ascending and transverse portions of the aortic arch can possibly be explained by the frequent involvement of the aortic valve by the inflammatory process, with its resulting insufficiency In this case, mechanical stress over the initial portion of the aortic wall is greatly increased by the association of elevated blood pressure with increased cardiac output The abundance of vasa vasorum in the first portion of the aorta is also considered by some as predisposing to the localization of spirochetes in this segment of the vessel

We have thus placed the vasa vasorum in the center of interest, admitting, of course, that the distribution of elastica and the ground substance and the tensile strength of the adventitia are significant, though perhaps not primary, factors in the development of aneurysms The nozzle effect of a proximal stenosis is often not detectable

CLINICAL FORMS

Saccular fusiform or diffuse enlargement of an arterial segment with or without a venous communication may be of congenital traumatic, inflam matory or degenerative (arteriosclerotic) origin Of these the congenital and traumatic aneurysms have already been discussed

Inflammatory Aneurysms

MYCOTIC ANEURYSMS Since subacute bacterial endocarditis is often efficiently controlled by antibiotics, surgeons encounter peripheral aneurysms in afebrile patients whose disease has been—at least temporarily—arrested Two iliac aneurysms and one axillary aneurysm in patients suffering from bacterial endocarditis have been excised on our service The inflammatory reaction around the sac and the histologic sections of the arterial wall leave little doubt that these dilatations are due to emboli to the vasa vasorum with resulting ischemia or necrosis of the wall Such aneurysms are usually ar terial but arteriovenous ones have also been noted More frequent are trau matic arteriovenous aneurysms which attract bacteria or their vegetations around the site of the fistula Excision of the arteriovenous fistula has cured the bacteremia [29]

Secondary infections in congenital traumatic and arteriosclerotic aneu rysms are not rare We have seen an unrecognized popliteal aneurysm be come purulent and rupture following a prostatectomy with a urine culture of *Staphylococcus aureus* The culture of the infected clot yielded a hemolytic staphylococcus Also aortic aneurysms often harbor abscesses in their wall and intestinal infections with periaortic glandular involvement may gradually spread into the wall of the aorta In the case of a well known midwestern surgeon, an amebic dysentery with secondary infection seemed the starting point of an aneurysm of the abdominal aorta Abscessed lymph nodes are not uncommon around abdominal aortic aneurysms

All forms of acute arteritis (degenerative necrotizing, exudative vegeta tive, proliferating and organizing)[30] may lead to a weakening, dilatation and elongation of the wall especially if they lead to swelling, splitting and frag mentation of the elastica Almost any acute infectious disease may produce this by direct invasion or by way of a hyperergic inflammation Thus in polyarteritis nodosa miliary aneurysms are common A necrotizing exuda tive arteritis in the vasa vasorum resulted in a fusiform aneurysm of the aorta [30] Rheumatic arteritis in the vasa vasorum resulting in destructive arteritis has been reported by Pirani and Bennett[31] (fig 254)

When the inflammatory process involves and denudes the intima, thrombosis is more apt to occur than aneurysm formation. In luetic arteritis the ground substance and the connective tissue show the most proliferation

LUETIC ANEURYSMS Of 547 patients suffering from cardiovascular syphilis 309 had aortic insufficiency 95 had aneurysms, 2 had coronary ostial stenosis and 141 exhibited syphilitic aortitis [32] While it is often stated

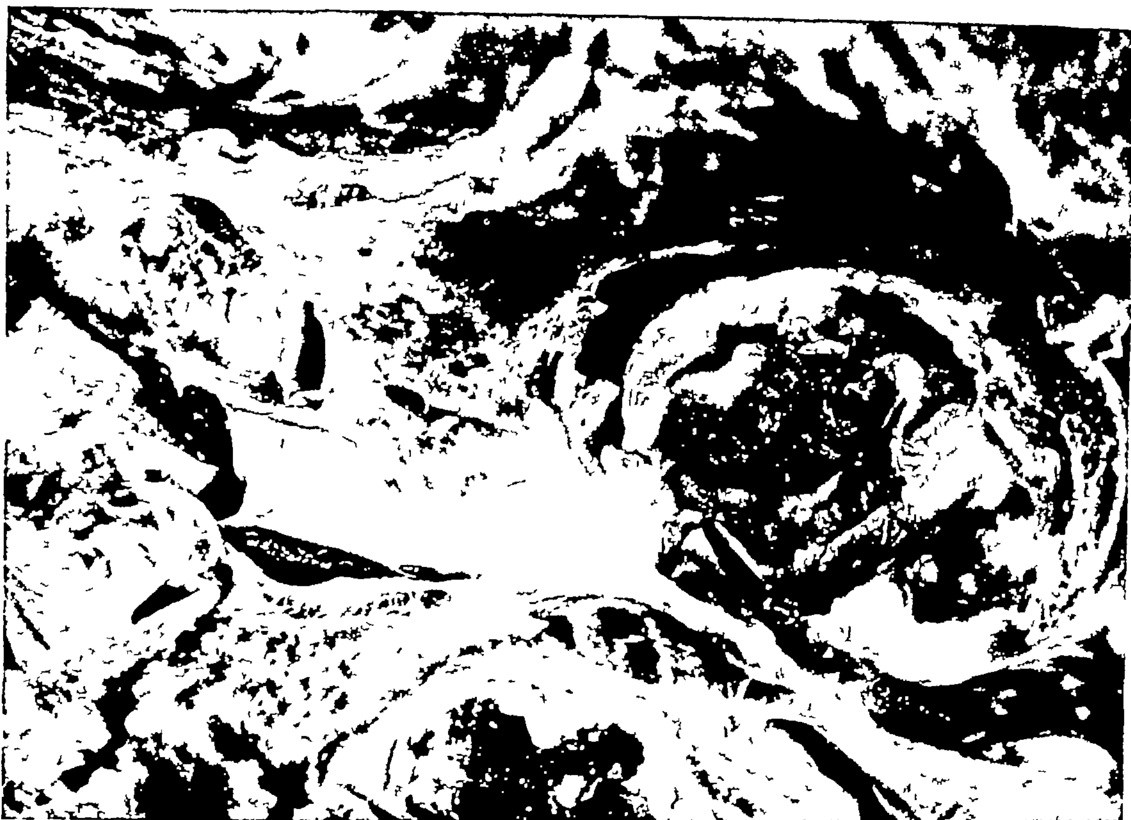


FIG 254 Mycotic aneurysm of the pulmonary artery (Courtesy of Dr C L Pirani)

that early vascular syphilis cannot be diagnosed accurately, the roentgen ray evidence of a widened aorta, the accentuated aortic second sound, the submanubrial dullness and a systolic murmur at the aortic area are important diagnostic leads

15 per cent of all such untreated patients developed aortic insufficiency or an aneurysm after an average of 3 5 years When arteriosclerosis or hypertension were added factors, the prognosis was worse

Against this natural history of syphilitic aortitis, the general prognosis of the adequately treated luetic aneurysm is favorable Rich and Webster[32] believe that 2 4 million units of penicillin as early treatment are adequate

Among aneurysms of the aorta, the thoracic lesions predominate (fig 255) Vertebral erosion, tracheobronchial obstruction and recurrent laryngeal paralysis produce characteristic symptoms (fig 256) In the abdomen, the aortic aneurysm is overwhelmingly arteriosclerotic and this will be discussed on page 394.

Luetic aneurysms may, of course, occur in other vessels of the body A large luetic femoral aneurysm occurring in a patient with general paresis and nitrogenous retention is shown in figure 257, a suspected luetic aneurysm of the popliteal area is seen in figure 258

With increasing prophylaxis and adequate early treatment of syphilis, the appearance of these large intrathoracic luetic aneurysms should decrease Because of penicillin, the number of patients with acute syphilis who receive adequate treatment has increased enormously Since this is usually curative treatment, it seems certain that the incidence of cardiovascular syphilis, a

late result of chronic infection will diminish greatly Even before the advent of penicillin syphilis morbidity and mortality showed marked reduction

Equally certain is the fact however that penicillin cannot alter the course of events once the complications of syphilitic aortitis such as aneurysm have appeared Penicillin can also produce febrile reactions or aneu



FIG 255



FIG 256

FIG 255 A luetic aneurysm of the transverse aortic arch and the innominate artery Surgical treatment was refused.

FIG 256. Mrs A G age 58 shows esophageal compression and deviation caused by luetic aneurysm



FIG 257 A luetic femoral aneurysm in a 52 year old Negro man dying of uremia.



FIG 258 Giant femoropopliteal aneurysm in a 72 year old woman with no evidence of peripheral arteriosclerosis, and a negative Wassermann reaction in the blood Lues was suspected but not proved

rysmal perforation (as in a personally observed case). A course of penicillin treatment before operative removal of a luetic aneurysm has been occasionally, but not routinely, carried out

POLYARTERITIS (PANARTERITIS) NODOSA The nodules in this disease are due to the formation of small aneurysms Microscopically, this is a necrotizing exudative arteritis Whether thrombosis, hemorrhage or aneurysm develops depends mostly on the distribution of the destruction of medial and elastic fibers Fatal hemorrhage from an intercostal artery into the pleural cavity or into the perirenal space has been reported [30] This allergic or hyperergic arteritis can also cause cerebral hemorrhage in patients who were labeled as having Buerger's disease [33]

Degenerative Aneurysms

ARTERIOSCLEROTIC ANEURYSMS These constitute today a large and steadily increasing percentage of the lesions seen at autopsy Better control of syphilis and increasing longevity of the population may be partly responsible for this shift in frequency The abdominal aneurysms run a long, asymptomatic course In Estes' report, 30 4 per cent of 102 patients had no symptoms referable to the aneurysm.[34] The scattered calcified plaques, the curvilinear, linear or laminated calcification, or simply the presence of a large

oval or spheroid soft tissue mass can be seen in a lateral roentgen film in 86 per cent of the cases[35] (fig. 259)

While some aneurysms may remain stationary or even asymptomatic most of them leak rupture or become thrombosed The slowly leaking abdominal aneurysm is a definite entity [37] It is not a dissecting aneurysm and creates massive organized fibrotic masses giving suspicion of retroperitoneal tumors Extensive fibrous induration of the pelvis and of the retroperitoneal tissues demonstrate the sac, but never show its extent because of the mural thrombus (fig 260) Neither the proximal extension of the sac above the renal artery nor its distal extension beyond the bifurcation has proved to be a limiting factor of operability and therefore aortograms need not be performed [36]

Rupture of the abdominal aorta occurred in 38 cases of abdominal aortic aneurysm collected from Canadian institutions by Copping.[38] The rupture was linear in most cases, measuring 2 8 cm on the average There was no dissection of layers and in three fourths of the cases the blood extravasated into the retroperitoneal fossa There was seldom massive blood in the peritoneal cavity although blood-stained free fluid was found in a number of cases The pain of a lower aortic rupture is agonizing and piercing, the patients may shriek in frenzy In one of our own cases a patient whose aneurysm had been wrapped and constricted with cellophane four years



FIG 259



FIG 260

FIG 259 Dilated calcified abdominal aorta in a lateral roentgen film. The patient was 76 years old and had no symptoms.

FIG 260. A saccular aneurysm of the abdominal aorta, starting well below the renal arteries. Note the width of the aorta proximal to the aneurysm, calling more for a distensible prosthesis than a frozen homologous graft.

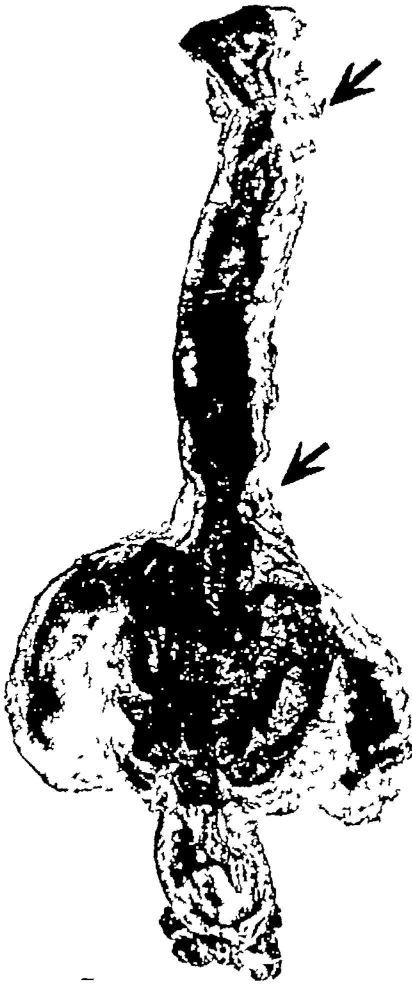


FIG 261 Acute thrombosis of an arteriosclerotic popliteal aneurysm with partially occluding atheromata in Hunter's canal The patient's foot and lower leg were gangrenous on entrance to the hospital and supracondylar amputation was necessary

previously, symptoms of rupture were interpreted by his local physician as being due to a ruptured appendix In other cases, coronary thrombosis, renal calculus and ruptured abdominal viscus were diagnosed It is important to realize that previous nonfatal episodes may have occurred, and evidence of sealed-off linear tears with an organized thrombosis capping them may be found It is well to regard anyone who has a known, palpable aneurysm and exhibits pain in the back, in the renal region or in the lower abdominal quadrants as having a threatening perforation, which calls for immediate definitive surgery In DeBakey's series, the mortality of resections of ruptured aneurysms was 33 per cent as against 8 per cent of the nonruptured ones [36]

Sites of arteriosclerotic aneurysms In addition to the thoracic and abdominal aorta, the popliteal artery is comparatively frequently involved, often bilaterally It is possible that the intermittent traumatic compression of the lower femoral artery in Hunter's canal may contribute to the development of popliteal aneurysms When these suddenly fill up with thrombi they are often confused with arterial embolism or thrombosis In figure 261 the thrombus extends high into the femoral artery and two atheromata are seen to produce a 50 per cent constriction of the artery at a typical location in Hunter's canal

Prior to occlusion or rupture of this aneurysm, and in cases where the

pulsating sac is doubtfully palpable in the obese individual huge oscillometric swings may clinch the diagnosis [39]

The elongation and tortuosity of the sclerotic carotid artery may give rise to the diagnosis of aneurysm Deterling[40] has recently summarized the literature and added a number of cases I have explored two hypertensive women with this syndrome which may produce vertigo and fall in blood pressure when the carotid sinus is stimulated

DISSECTING ANEURYSMS There is a striking association of deficiencies of the aortic wall with congenital vascular anomalies such as coarctation of the aorta patent ductus arteriosus septal defects bicuspid aortic valves or Marfan s syndrome In 141 cases of dissecting intramural hemorrhage in persons under 40 31 per cent exhibited some degree of congenital aortic narrowing.[41] The absence of elastic fibers and the mucocystic degeneration could be seen at considerable distance from the dissecting segment

Although hypertension and sudden strain have been emphasized in the older literature as etiologic factors in the production of dissecting aneurysm, the presence of a degenerative process in the media seems to be the basic factor Following total thyroidectomy for cardiac decompensation Kountz[42] reported four cases of advanced medial degeneration three of whom died of rupture The central position of the elastic tissue and its metabolic relationship with the basophilic ground substance have not been sufficiently explored [43]

Gore has recently called attention to the rupture of newly formed vasa vasorum in the media the intramural apoplexy seems primary and initiates the hemorrhagic dissection According to Gore the intimal laceration is secondary to the extravasation of blood in the media He noted 23 cases of extensive intramural hemorrhage communicating neither with the lumen of the aorta nor with the adventitia.[44] He also reported 72 cases from the Armed Forces Institute of Pathology [45] The medial degeneration was of two types In the first the elastica seemed to be involved and this was more often observed in persons under 40 years of age In the second type muscular involvement was more prominent This occurred in the older group Naturally a combination of both types occurred in an intermediate age group

Interestingly a rechannelization of the intramural tract back to the parent lumen may occur either through another distal tear of the intima or through the natural ostia of the intercostal or lumbar vessels [45]

I have quoted the studies of Gore[44, 45] because he injects an interesting line of thought into the etiology of dissecting aneurysms As a matter of fact, this thought had been expressed by Krukenberg[46] and others many years ago The origin of the medial degeneration with the appearance of mucin will need much further study along lines of nutritional and vitamin research

Dissecting aneurysms may readily give rise to erroneous diagnoses The severe chest or back pain may simulate coronary thrombosis the dissection clear down to the iliac bifurcation may appear as an aortic or iliac embolus the dissection may also be painless In the most severe form dissection is so rapid that perforation occurs into the pericardium, mediastinum or

peritoneum, and the patient arrives at the hospital in a moribund state, only to die in a few hours There is, however, a subacute type in which gradual dissection occurs over a period of days or weeks, finally ending in rupture and death And, finally, there is an occasional self-healing process in which the false route re-enters the original lumen of the aorta and may even become covered with endothelium so that a double-barrelled aorta develops. Occasionally one can obtain a peculiar aortogram, such as that obtained on the cardiovascular service of St Luke's Hospital [47] In this 65 year old hypertensive man a painless dissection occurred, with loss of pulsations in the right leg and arm He died suddenly from a rupture into the pericardium on the fourth day of his illness A recent dissection of the entire aorta with obstruction of the right iliac artery was found at autopsy (fig 262) A dissection of aortic layers may be produced by an ill placed needle during an aortogram, giving a wide smooth-contoured aorta with no branches (fig 263) The severe thoracico-abdominal pain of dissection is produced No permanent ill effects have been observed in three such cases

DIAGNOSIS OF ANEURYSMS

Aneurysms, or saccular or fusiform dilatations of an artery, may appear in any blood vessel of the body Here we will briefly describe those locations which are most frequently encountered and which give characteristic clinical symptoms

Pulsatile swelling along the course of an artery is always highly suspicious of an aneurysm, but, obviously, transmitted normal arterial pulsation under a mass of glands, tumors or abscesses is always to be considered. Thoracic aneurysms are well visualized by a chest film, but mediastinal lymph glands and tumors need to be excluded The old dictum that a mediastinal mass with a negative Wassermann reaction is not an aneurysm certainly does not hold One may have to resort to an angiocardiogram to get a definite diagnosis (fig 57) In a case of a rhabdomyosarcoma of the thigh (fig 46), a normal femoral artery was seen to course through a pulsating mass in a femoral arteriogram

Conversely, a pulsating mass in the axilla, groin or femoropopliteal region is still mistaken for an abscess and thoughtlessly incised I have personally observed such pulsating hematomata being entered by physicians and even by a surgical resident, leading to profuse hemorrhage and necessitating prompt intervention on the artery

Aneurysms tend to enlarge at an unpredictable rate and produce pressure on neighboring structures Erosion of vertebral bodies are caused by thoracic aneurysms of the aorta, and less frequently by the abdominal ones Pressure on the posterior nerve roots and posterior root ganglia produce an intractable, segmental type of pain, the esophagus and trachea are frequently misplaced and obstructed Carotid, axillary, femoral and popliteal aneurysms press on neighboring nerves resulting in characteristic radiation Particularly impressive is the pain in the heel in popliteal aneurysms caused by pressure



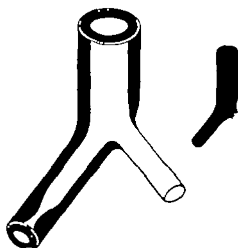


FIG 262. The aorta and a wide right iliac artery are visualized, although the iliac pulse could not be felt the nonvisualizing left iliac artery however exhibited a bounding pulse The dark areas visualized in the roentgen film and the diagram are due to the injection being made into a false channel. (Lary B G and Davis, J A Paradoxical Aortogram in Dissecting Aortic Aneurysm Ann. Surg. *142* 304 1955)



A



B

FIG 263 (A) Dissection of the abdominal aorta by an ill placed needle into the layers of the aorta (B) a roentgenogram taken six hours later The opaque substance is spreading cephalad In 24 hours the dye had disappeared.

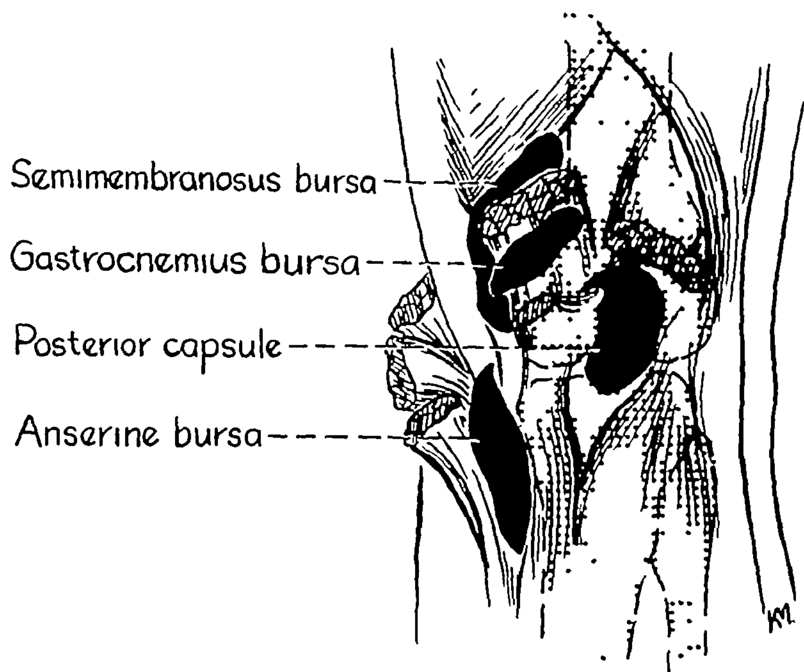


FIG 264 The bursae around the posterior surface of the knee joint which may show transmitted pulsation The popliteal bursa in the midline, communicating with the knee joint through the posterior capsule, may compress the popliteal nerve and vein and be mistaken for a popliteal aneurysm

on the popliteal nerve This may be one of the first symptoms of a small aneurysm which may be difficult, if not impossible, to palpate in a popliteal fossa that is well padded with fat and often containing bursae, which themselves may exhibit a transmitted pulsation of a widened, sclerotic, but not aneurysmatic, artery (fig 264) Pressure on the concomitant veins is a frequent finding Thus, the aneurysms of the ascending and transverse arch of the aorta produce an obstruction of the superior vena cava and its branches Abdominal aneurysms, since they have enough room to expand, do not obstruct the inferior vena cava, although they are often firmly plastered down to it Axillary, femoral and popliteal aneurysms frequently retard or obstruct venous return

Tortuous, elongated or sclerotic arteries may be mistaken for aneurysms Reference has already been made to the tortuous carotid artery mistaken for an aneurysm (p 397) [40] The elongated, sclerotic, abdominal aorta in a thin individual is tender to pressure and is a fairly frequent "aneurysm" to students, residents and even resection-happy surgeons The horseshoe kidney is particularly apt to thrust an elongated aorta forward and may even be associated with an aneurysm

Auscultation over the pulsating mass reveals a systolic murmur, and if the murmur is a to and fro, continuous, machinery-like murmur, an arteriovenous communication must be present However, the presence of a systolic murmur may signify arterial stenosis with turbulence and is not characteristic of an aneurysm In fact, as pointed out by Edwards and Levine,[48] systematic auscultation of vessels at or above arterial compression will lead to the diagnosis of arterial stenosis As pointed out in chapter 10 under periph-

eral arteriosclerosis (p 103) one can visualize such a bruit with a stethogram

Certain deep-seated aneurysms are not accessible to palpation or auscultation In cerebral aneurysms, which are outside the scope of this monograph cerebral angiograms give definite information [49] Occasionally the patient hears a buzzing sound or the examiner may hear it with a stethoscope, but the angiogram has superceded the occasional value of such findings Aneurysms of the chest and abdomen can be heard or can be visualized by roentgen ray An arteriovenous aneurysm between a branch of the superior mesenteric artery and vein—following a resection of the ilium—could be well heard through a thin abdominal wall The aneurysms of the splenic, hepatic and renal arteries cannot be heard but may be picked up by aortography Most of them are found accidentally at laparotomies undertaken for undiagnosed abdominal hemorrhage Generally speaking, these visceral aneurysms cause symptoms of pain because of the stretching of the wall and rupture with sudden hemorrhage or interference with the vascularity of the organ which they supply with blood

Specifically aneurysms of the splenic artery have created much interest since in 1881 President James Garfield died from a ruptured splenic aneurysm two months after being shot in the abdomen [50] Owens and Coffey[51] analyzed 198 cases with the addition of six cases of their own and pointed to the pathogenetic factors of congenital vascular defects trauma embolism and arteriosclerosis Pregnancy was noted in one half of the female patients of child bearing age Evidence of portal hypertension was found in 20 per cent of the collected cases and one of these operated on by Warren H Cole at the Research and Educational Hospitals of the University of Illinois appeared to be due to a congenital arteriovenous fistula for which splenic artery ligation followed by splenectomy was performed In case of severe hemorrhagic shock proximal ligation may be carried out Roughly one half of these aneurysms rupture

For other visceral aneurysms, such as those of the hepatic, superior mesenteric or renal arteries resection of the aneurysm with restoration of continuity needs to be performed since the organs served are not expendable an exception is the kidney although even here an effort to save the involved side is worth while

TREATMENT OF ANEURYSMS

Many of our previous attempts to obliterate aneurysmal sacs or to excise them without restoration of continuity are either obsolete at this writing or need to be limited to a few exceptional cases Since aneurysms by their own expansile natural history either rupture or thrombose, their recognition is an indication for their elimination The overwhelming experience of Michael E DeBakey and his associates in this field[36] leads me to conclude that all three forms of aneurysm of the aorta (namely the saccular the fusiform and the dissecting) are amenable to surgery and that with growing

experience the mortality of these formidable lesions can be successfully reduced. The mortality here depends on such factors as age, the existence of hypertension, the presence of cardiovascular or renal disease and the presence or absence of rupture at the time of operation Location, of course, is a most important factor, the resection of abdominal aortic aneurysms without noticeable heart disease giving a 2 per cent mortality Since in this monograph we are not concerned with thoracic aneurysms, it will just be mentioned that the use of bypasses and extracorporeal shunts has given DeBakey and his associates the best results, and that hypothermia with its attendant side effects is gradually being de-emphasized [52]

Unfortunately, many patients with abdominal aortic aneurysms arrive at the hospital after the aneurysms have ruptured, without the patient having had any previous knowledge of their existence Javid and his associates[53] have reported a 50 per cent mortality from our institution in 1955, and this figure still holds at this writing. The high mortality is, of course, the greatest incentive for the removal of these masses before they rupture

The techniques of dealing with aneurysms at different locations of the body will be described in chapter 21, Surgical Procedures Here only the principles of treatment will be outlined

(1) Excision of the Aneurysmal Mass with its Feeding Vessels This is our practice in congenital vascular anomalies, but in the traumatic aneurysms and arteriovenous fistulae, every attempt is made to restore arterial continuity A special group headed by Daniel C. Elkin examined over 800 veterans with arterial injuries incurred during World War II (chapter 8, Arterial Injuries) [54] In spite of sympathectomies added to aneurysmal excision, the exercise tolerance of these patients has not improved, although their sensitivity to cold has been ameliorated [55] Naturally, smaller vessels whose ligature is inconsequential need not and most often cannot be restored in continuity; such vessels are the radial, ulnar, anterior or posterior tibial arteries and small visceral vessels Excision of the popliteal aneurysm, with and without sympathectomy, is usually tolerated by the patient without a loss of limb, however, exercise tolerance is poor and here again restoration of continuity is to be preferred.

(2) Proximal Ligation of the Artery In congenital arteriovenous fistulae this procedure may be followed by gangrene (fig 95) In traumatic, luetic and arteriosclerotic aneurysms a clotting of the sac may be produced, but such a procedure has only historical interest today Severe ischemia and distal propagation of the clot are great hazards With the exception of carotid artery ligations for aneurysms of the brain in certain well selected cases,[56] this method is to be condemned Complete ligation of the aorta in one or several stages has been tried in the past, but erosion of the wall, aneurysm proximal to the ligature and ischemic damage to the cord and to the lower extremities have made this method obsolete and unjustifiable

(3) Partial Proximal Constriction. Blakemore[8] has measured blood flow below the banding, and has emphasized the harmful effects of anything less than 75 per cent constriction This finding, together with the

more recent techniques for eliminating aneurysms precludes further use of these interesting and painstaking attempts to obtain precisely the right amount of stenosis

(4) LIGATION OF THE PROXIMAL VEIN IN ARTERIOVENOUS FISTULAE As an emergency procedure in cardiac failure, this method may restore compensation so that definitive therapy can be undertaken (cited in 2)

(5) WIRING OR WRAPPING WITH CELLOPHANE. An analysis of the methods of wiring and wrapping with cellophane alone or combined with partial constriction has been made in our clinic with Marshall[57] (fig 265) We have surrounded the sac with cutis graft and steel mesh and have wired a great many aneurysms, both thoracic and abdominal in the past (figs 266 267) It can be said with assurance that these methods have been superceded, and even though I have spent much time in studying and carrying out these procedures, there is no more justification for their use At best a complete aortic occlusion is produced with all its functional disability (fig 268)

(6) PROXIMAL LIGATION OF THE ARTERY COMBINED WITH A BYPASS This may be occasionally indicated in aged, debilitated cardiac patients a large axillary aneurysm of 13 years duration in a syphilitic and arteriosclerotic patient with marked cardiac irritability was managed in this fashion [58] The cephalic vein used for the shunt remained open for at least two years and the mass gradually became impalpable (fig. 269)

(7) EXCISION OF THE ANEURYSMAL SAC WITH RESTORATION OF CONTINUITY Whether the best graft for this purpose is a homologous frozen arterial graft or a plastic prothesis is still open to question although the present trend is toward the plastic prosthesis This should be flexible slightly porous nonirritating to the tissues and noncarcinogenic and should exhibit a two-way stretch Wherever the aneurysm is, this treatment applies, and unless the patient s cardiovascular renal status is poor it can and should be done with steadily decreasing mortality and morbidity In addition to aneurysms

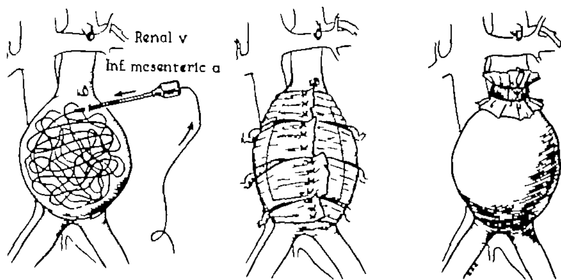


FIG 265 Wiring, wrapping and partial constriction alone and in combination have been carried out in our clinic in the past.[57] These methods are not in use today



FIG 266



FIG 267

FIG 266 A wired luetic aneurysm of the ascending aorta The patient was relieved of pain and pressure symptoms for two years He died of heart failure

FIG 267 Abdominal aneurysm wired with 20 feet of new-silver wire The patient developed an embolus to the left foot and lost one toe Generally speaking, wiring produced longer palliation than wrapping with reactive cellophane



FIG 268 A large arteriosclerotic aneurysm, wrapped with reactive cellophane The patient died two years later with cardiac failure Huge connective tissue reaction and an old occluding thrombosis of the aortic lumen are seen (de Takats and Reynolds Surgical Treatment of Aneurysms of the Abdominal Aorta Surgery, *21* 443, 1947)

of the aorta, iliac femoral, popliteal innominate subclavian axillary and brachial aneurysms can be so repaired The percentage of patency in the grafts is high about 90 to 95 per cent and secondary hemorrhages from the suture line are infrequent although they do occur notably after the use of plastic prostheses Outside of the aneurysms of the ascending aorta and trans verse arch mortality has become less and less of a problem [36]

(8) RESECTION OF A SACCULAR ANEURYSM WITH LATERAL SUTURE. This has been especially advocated by Bahnson[59] and is an excellent benign procedure particularly suitable for luetic saccular aneurysms of the thoracic aorta The luetic arterial wall sews well but the patient may have to be pre loaded with penicillin

(9) VEIN INLAY GRAFT OR PARTIAL RESECTION OF THE ANEURYSM WITH SUTURE. These are two other methods which may have occasional use (fig. 270) Autogenous veins are unfortunately seldom expendable except dilated saphenous veins furthermore the wall of the sac is seldom suitable for safe suturing. Nevertheless, the principles of these methods may be applied in cases where no other material is available

(10) TREATMENT OF DISSECTING ANEURYSMS For dissecting aneu rysms, a condition which is rapidly fatal in 75 to 90 per cent of the cases Michael DeBakey and his associates have devised two distinct procedures [60]

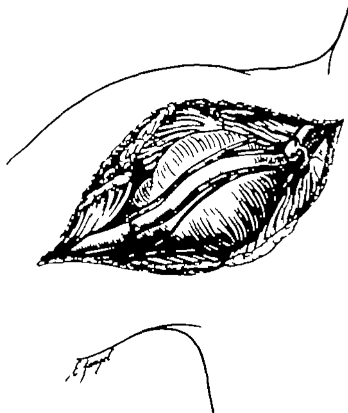


FIG. 269 An axillary aneurysm in a cardiac, luetic and arteriosclerotic patient, bound down to the brachial plexus. Instead of removing the mass, the blood flow was sidetracked by sectioning the proximal and distal ends of the artery and connecting the two stumps with a graft taken from the cephalic vein. The aneurysm clotted, shrank and became impalpable. The pulse below the aneurysm was restored. (Cl. McK St. Luke's Hospital, 1953 ■)

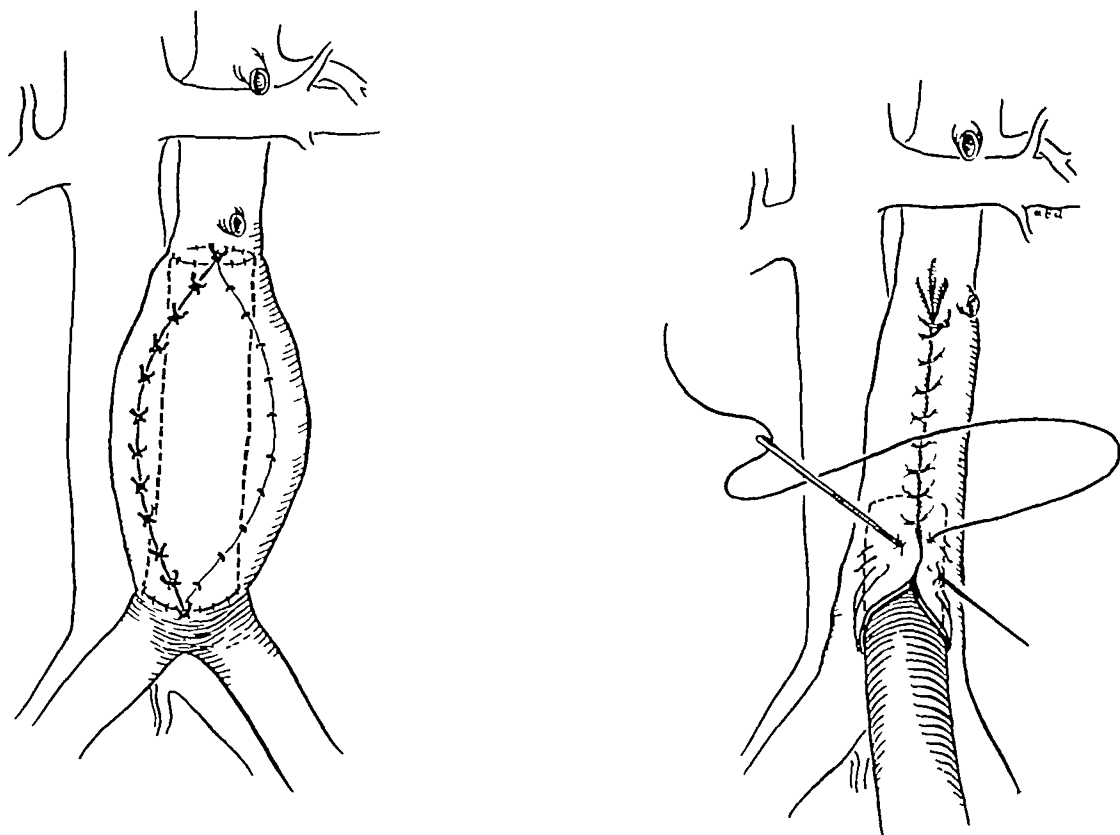


FIG 270 On the left a vein inlay graft is shown, on the right the protruding sac has been resected and the lumen reconstructed over a large rubber catheter (de Takats and Marshall Surgical Treatment of Arteriosclerotic Aneurysms of the Abdominal Aorta Arch Surg, *64* 307, 1952)

When the dissection begins in the ascending part of the thoracic arch, a re-entry passage is created by excising a wedge-shaped segment from the inner, intimal layer The false passage below, which may be as low as the diaphragm, is obliterated by approximating the outer and inner layers The operation is then completed by an end to end anastomosis If the lesion is fairly well localized and arises at or below the left subclavian artery, the segment of the aorta involved in the dissection can be excised, the false passage obliterated and aortic continuity restored by end to end anastomosis or by the insertion of a graft

Of a total of 16 cases reported, 12 patients were treated by excision with lateral aortorrhaphy, end to end anastomosis or insertion of a homograft, with two deaths The other four patients had a re-entry passage created, and of these two died The survivors have remained symptom-free for as long as one and a half years [61]

Resection of the abdominal aorta with replacement and the procedures for the treatment of dissecting aneurysms will be illustrated in chapter 21, Surgical Procedures

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CHAPTER 16

NEUROVASCULAR LESIONS OF THE EXTREMITIES

1 RAYNAUD'S SYNDROME

IT HAS BEEN CUSTOMARY TO SEPARATE EVER SINCE THE INCISIVE COMMENTS OF Sir Jonathan Hutchinson [1] Raynaud s disease a primary idiopathic symmetrical vessel spasm of the digital arteries from Raynaud s phenomena, which are due to a multiplicity of organic lesions more or less obvious at the initial examination It will be noted that the term *Raynaud's syndrome* is used here since with growing experience the primary essential idiopathic forms seem to dwindle and the digital syncope cyanosis and rubor accompanied by numbness and followed by burning reveal themselves as a set of symptoms of a variety of disorders

In all fairness to Raynaud whose report in 1862 contained 25 case histories [2] his thesis was occupied mainly in disputing Baron Dupuytren s contention that all cases showing digital color changes were due to organic arterial occlusion As pointed out by Jepson [3] each side was partly correct, as in many historical disputes Our recent understanding of the many then unknown neurologic, hematologic and connective tissue disorders leads one to believe that Dupuytren was closer to the truth than Raynaud

Thus in this chapter I shall describe this symptom with the obvious acknowledgment that its underlying cause is frequently obscure and often unknown.

Intermittent attacks of pallor followed by cyanosis brought on by exposure to cold or emotion characterize Raynaud s syndrome In this era of functional pathology one might readily believe that the repeated attacks of digital arterial spasm finally lead to proliferative endarteritic thrombosis There is nothing to refute the notion however that early vascular disease responsible for the cold sensitivity progresses to total occlusion and that the increasing attacks of syncope simply mean the advance of digital arterial stenosis played upon by normal vasomotor fluctuations

PATHOPHYSIOLOGIC DATA

In the uncomplicated early Raynaud s disease the absence of organic digital arterial lesions has always been stressed based on the finding that reflex vasodilatation or peripheral nerve block would heat up the finger to a

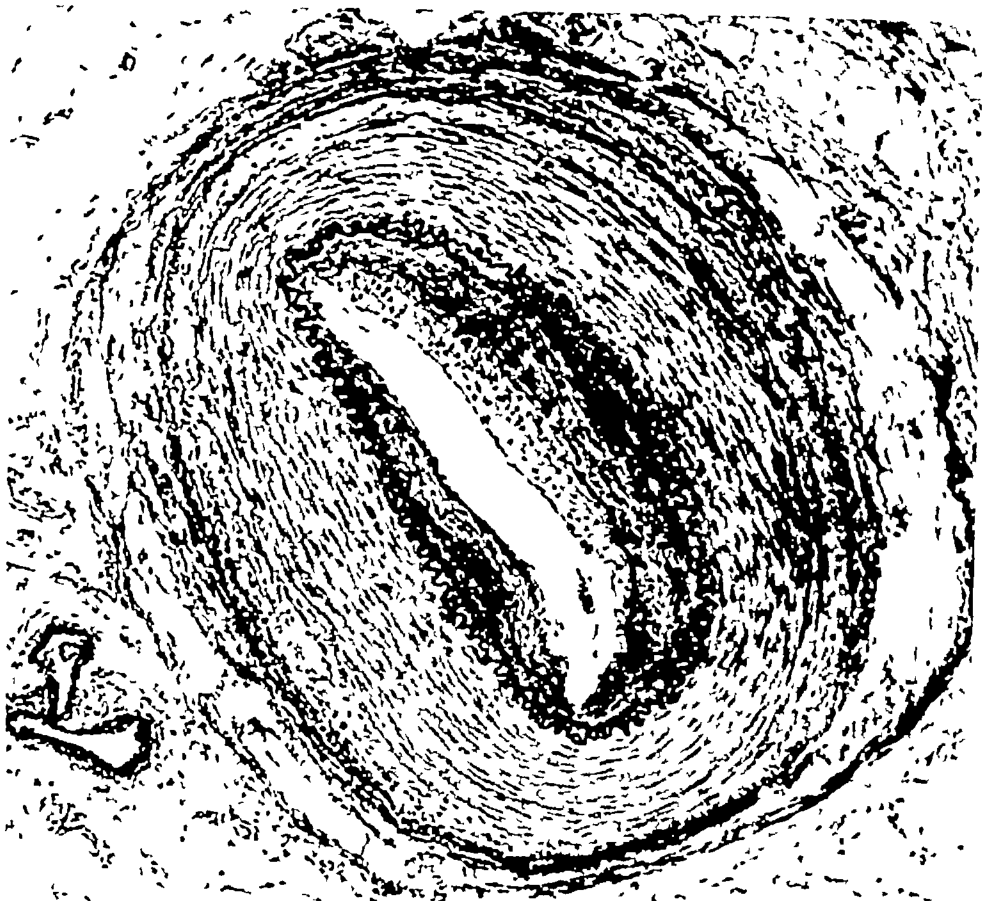


FIG 271 Digital artery of a 23 year old woman suffering from uncomplicated, "primary" Raynaud's disease and dying of an intercurrent disease Note the intimal proliferation, some splitting of the internal elastic membrane and thickening of the muscular coat (Courtesy of Dr R M Goetz and the Williams and Wilkins Co [4])

normal vasodilatation level When the pulse volume is studied with digital plethysmography after reflex heating for 30 minutes, the blood flow was found to be subnormal in at least one third of the cases [4] Goetz published a report of a section of the digital artery done on a 23 year old woman suffering from Raynaud's phenomena, who died suddenly of an intercurrent disease [4] While the radial artery was completely normal, the digital vessels showed marked proliferation of the intima, splitting of the internal elastic membrane and thickening of the muscular coat (fig 271) While 66 per cent of his 120 patients studied with Raynaud's phenomena showed a normal response of the plethysmographic pulse wave to indirect heating, these "normal" flows may be due to the inability of this method to register early subintimal changes Perhaps not all of the patients had Raynaud's syndrome

This is not to say, however, that sudden neurogenic or hormonal impulses cannot lead to digital pallor There are a whole lot of neurologic disorders, including syringomyelia, anterior poliomyelitis and spinal and cerebral tumors, and also peripheral irritative lesions seen with a cervical rib and the thoracic outlet syndrome which may produce intermittent digital spasm on exposure to cold In a personally observed case, an anterior scalenotomy was followed by Raynaud's phenomena in a patient who had never had any Raynaud's phenomena before On excising a thick scar which surrounded

the stellate ganglion the vasospastic attacks disappeared One also sees scalene nodes thickened apical pleura or adhesive mediastinitis give rise to vasospasm, not to speak of the frequent radiculitis due to cervical osteoarthrosis It should be emphasized however that such lesions hardly ever cause the *intermittent* vasospasm being discussed here

While Raynaud concentrated his attention on an increased vasomotor tone Sir Thomas Lewis[5] concluded that a local fault, an abnormal sensitivity of the digital arteries to cold is the basic factor in the late cases this will lead to structural changes, such as intimal thickening and thrombosis [6]

More recently the role of agglutinated red cells[7] or precipitated gamma globulins [8] both brought on by the effect of cold in sensitized patients has been emphasized When such a red cell aggregrates or when gelled proteins cause an obstruction in the smaller vessels it is likely that the vessel wall would hug this plug this is what happens in an arterial embolism Schwartz and Jager[9] produced a cutaneous ischemia surrounded by a halo of erythema by placing an icebag for five minutes on the patient s chest they thought they could detect cryoglobulinemia in this way and differentiate it from Raynaud s disease

In our clinic, cold agglutinins and cryoglobulins have been routinely looked for and the conjunctival vessels have been studied by a slit lamp with the cooperation of ophthalmologists With the exception of an occasional positive report of sludging in conjunctival vessels when exposed to an ice cold drop of physiologic saline solution,[10] these determinations yielded negative results in Raynaud s syndrome and their clinical significance at this time is not apparent As will be pointed out later acute hemagglutinins following atypical pneumonia may be found in patients exhibiting acrocyanosis [11]

The intermittent attack of digital arterial spasm may thus occur in early lesions of the vessel wall and in patients who show cell clumping or protein gelling as a result of exposure to cold but also as a result of neurogenic stimuli Perhaps the best example of this is the Raynaud s phenomenon occurring after dorsolumbar sympathectomy for hypertension [12] the increase in sympathetic activity in the nondenervated upper extremity is apparent and is accompanied by excessive sweating

Whether increased sensitivity of the vessel wall to epinephrine and norepinephrine plays a role is highly conjectural certainly the combination of a pheochromocytoma with Raynaud s syndrome is not known. On the other hand spontaneous or insulin induced hypoglycemia, which activates the adrenal medulla can bring on digital pallor

Thus the etiology and pathophysiology of Raynaud s syndrome is not clear and probably a multiplicity of factors is involved in its production

Clinical Picture

Attacks of pallor cyanosis and rubor occur and a triphasic reaction may occasionally be seen in young children But in these there is certain to be

an allergic vasculitis secondary to some severe infection, such as the meningococcus or streptococcus produces In chapter 9, Arterial Inflammation, cases have already been mentioned which come to symmetrical gangrene. The more usual picture is that of the thin, asthenic, anemic, hypometabolic young woman who does fairly well in a warm room, but in whom a critical temperature of 18° C (65° F) will precipitate an attack Such women have thin, long fingers and, should they also show hyperhidrosis, the thermoregulatory heat loss is greater through such areas. Maddock and Coller emphasized the fact that the extremities make up 65 per cent of the body surface,[13] and, of course, the arteriovenous shunts which open on response to cooling (chapter 2, Vascular Shunts) are numerous in the fingertips It may be quickly stated here that, while obviously desirable, neither correction of the hypometabolism nor of the anemia helps these vasomotor phenomena

Such patients do better while they are pregnant One patient requested the Red Cross to bring her husband back from England during World War II when she overheard in class that pregnancy influences Raynaud's phenomena favorably Actually, our clinic has never observed favorable results from estrogenic substances administered for Raynaud's syndrome, whether given by mouth or implanted as pellets Herrmann and McGrath studied a group of patients with arterial deficiency and superimposed vasospasm who were given parenteral estrogens No clear-cut results were obtained [14] Nevertheless, an endocrine disturbance at and after the menopause does bring on Jepson's "middle-aged female syndrome"[3] in women who have not had Raynaud's phenomena before, who have no evidence of arteriosclerosis or any other vascular disease and who may gradually develop sclerodactylia, a more advanced form of Raynaud's syndrome having no connection with scleroderma

Other patients, and these constitute the majority, exhibit the Raynaud's syndrome as part of diffuse collagen disease, the varieties of which have been discussed before (pp 183–194) This may be a scleroderma, a lupus erythematosus, a dermatomyositis or even a rheumatoid arthritis In young men, thromboangiitis is to be suspected, and in elderly people one sees digital or often aortic-subclavian atheromatosis; the latter gives differences in blood pressure and in intensity of the radial pulse or definite changes in skin temperature, especially after exercise

The appearance of pulmonary hypertension in three patients who have had Raynaud's phenomena in the hands[15] makes one wonder whether the late pulmonary fibrosis seen in Raynaud's disease and scleroderma are not primarily on a vasospastic basis The hyperplastic arteriosclerosis may be the end result of arteriolar spasm, but also may be due to organic disease, possibly an allergic vasculitis

Many of our patients exhibiting Raynaud's syndrome show an allergic diathesis, and a competent survey of their allergies is occasionally, though not very often, of some benefit

Another group of patients, who will be discussed with the vasoncuropathies, such as are due to chilling, compression and vibration (p 426), often

show Raynaud s phenomena however there is no reason to discuss them here in detail since their obvious etiology will not lead to any diagnostic confusion

Diagnosis

The criteria on which the diagnosis of Raynaud s disease is usually based are those of Allen and Brown (literature cited in Allen Barker and Hines[16]) They require (1) episodes of Raynaud s phenomenon excited by cold or emotion (2) bilaterality of the phenomena (3) absence of gangrene, or if it is present its limitation to minimal grades of cutaneous gangrene (4) absence of any primary disease responsible for these phenomena and (5) symptoms of two years or longer duration

Of course the difficulty of adhering to such criteria is that the primary disease may be *many years* in becoming clinically manifest The closer one looks and the longer one follows these criteria, the more they are apt to become a symptom and not a disease Even though the astonishing number of 198 male patients (23 per cent of the total group) were diagnosed as having Raynaud s disease or Raynaud s phenomena by the Mayo Clinic,[17] the original diagnosis seemed adequate in only 34 out of 100 of these patients Undoubtedly the diagnosis will rarely have to be made and then with utmost caution

When the organic process in the digital arteries progresses to thrombosis, trophic phenomena develop The fingers lose their fat pads, the terminal phalanx tapers off to a point and the nails stop growing and become brittle, showing a concave configuration. Small ulcers develop which heal slowly if at all leaving stellate scars at the tips of the fingers

One does not see in a true Raynaud s syndrome any massive gangrene of digits, hand or foot unless an additional factor supervenes The application of heat chemicals notably phenolized products, or some infection may produce edema lymphangitis or ascending thrombosis all of which cause critical ischemia in a poorly vascularized digit The present tendency to treat ulcerated or gangrenous areas without dressings and expose them to the drying properties of the air current has much to do with secondary infections in this situation

Treatment

Drugs, physical therapy and surgical procedures will be discussed in the management of Raynaud s syndrome with special emphasis on the indications and proper timing of these methods

Drugs

The following drugs have been advocated and found to be useless in our hands in the treatment of Raynaud s syndrome Prostigmin, Mecholyl by

iontophoresis, Padutin, Depropanex, papaverine and its derivatives, Cyclospasmol, Priscoline, the methonium compounds and Hydergine Of these, Priscoline may warm up cold hands, and constipated patients may find a welcome change in bowel habits When the drug is given in effective doses, it produces diarrhea, gooseflesh, nausea and heartburn

Locally, 2 per cent nitroglycerin ointment has been tried [18] Headache develops in almost all cases[19] and the results are not too impressive. I have observed two patients in whom slight relief was obtained, but one can produce an attack while the fingers are well anointed

In women whose attacks are definitely triggered or aggravated by menstruation, large doses of estrone, as much as 45,000 international units, given intramuscularly seem to be beneficial [20]

Of the vitamins, B_{12} in large doses seems to help the numbness and tingling, it possibly affects a peripheral neuritis causing antidromic vasodilatation (p 21) Roniacol, a derivative of nicotinic acid, may warm up the fingers between attacks so that the critical temperature at which the attack occurs is raised This is our favorite drug in chronic occlusive arterial disease, although it acts more on the blush areas than on opening the glomus

All these drugs and a legion of others may be tried in the mild cases, since if these do not show progression surgery would not be advisable. Actually, however, they have minimal if any influence on the disease, but they act as a simple form of psychotherapy which these people truly need

Psychotherapy

Our form of psychotherapy, which is employed before any operative procedure is undertaken, is to have the patients interviewed by a psychiatric social service worker The unloading of a long social history and background onto the worker has a beneficial effect on the patient Should a severe psychoneurosis be detected, both the patient and the surgeon are protected from the performance of any surgical procedure The patient is then referred to a psychiatrist. Just as in the case of hypertension, one never sees a patient with Raynaud's syndrome have fewer attacks or milder ones after psychotherapy, but their attitude toward the disease improves This can often be accomplished by simple and repeated reassurance Most patients have a deathly fear of losing both hands or feet and becoming completely incapacitated Dictionaries and newspaper columns are of no help

Physical Methods

Every patient suffering from Raynaud's syndrome has a critical temperature at which the digital vessels go into spasm Keeping the extremities and the whole body warm with gloves, warm clothing and hot alcoholic drinks, and moving to a warm climate are of some benefit It must be remembered that even if the patient moved to the Florida Keys, he would still have to be continuously exposed to an environmental temperature of 85 F. or more

before vasomotor control would be completely abolished Actually patients who have moved south for any form of vasospastic or chronic circulatory disorder are frequently disappointed The humidity and the fluctuations of temperature affect them adversely and the underlying disorder—unfortunately most frequently a collagen disease—marches on independently of climate or reassurance

Surgical Treatment

It is a curious fact that internists (Allen Barker and Hines[16]) are more impressed with the effects of surgical treatment than are the surgeons who believe that incomplete denervation and regeneration dog their efforts to obtain good results following sympathectomy for Raynaud s disease in the upper extremities [21] During the course of years our clinic has gone through periods of various operative procedures and of various indications for them Since both factors have a great deal to do with the success or failure of sympathectomy for Raynaud s syndrome these two points deserve some discussion

Between the years 1928 and 1936 we used the anterior approach of Gask and later moved on to Smithwick s dorsal preganglionic sympathectomy [22] combined with root section We then stopped doing root sections, but continued the extended cervicodorsal sympathectomies the chain being cut above the stellate and below the third dorsal ganglion through the anterior supraclavicular approach Finally the axillary transthoracic approach of Atkins was adopted,[23] and this is now our standard procedure The chain is cut at or just below the stellate ganglion and below the fourth dorsal segment Palumbo[24] has recently advocated a separation of the first dorsal ganglion with its white ramus from the body of the stellate ganglion he believes that this can be accomplished without producing a Horner s syndrome

Several points need emphasis regarding the necessary extent of sympathectomy for the upper extremities (1) As shown by Ray and his co-workers [25] sympathetic fibers in the first thoracic anterior root are present in 1 out of 16 cases in which the roots were electrically stimulated at laminectomy While I originally advocated the extension of sympathectomy to above the stellate and intermediate ganglia,[22] later experience of others and our own showed that the Horner s syndrome and the stuffy nose are definite drawbacks and that there is not a greater percentage of success with this extended cervicodorsal operation (2) The emphasis on preganglionic versus postganglionic section is unnecessary since epinephrine sensitivity while more pronounced after postganglionic section has nothing to do with the recurrence of Raynaud s phenomena [26] On the other hand preganglionic section carries a larger percentage of incomplete denervations since it removes no ganglia [27] (3) The operation preferred in our clinic at present is thoracic ganglionectomy from the second to below the fourth ganglia Should recurrence of symptoms occur the intermediate and stellate ganglia are re

moved by an anterior approach, we have carried out this procedure several times, with the improvement in symptoms for a few months followed by a recurrence For a certain number of upper extremities, complete sympathetic denervation is impossible, and this is one of the most potent causes of failure According to Smithwick and his co-workers,[27] in 25 upper extremities which were followed from 5 to 15 years and which showed incomplete denervation, 11 showed good, 10 showed fair and 4 showed poor results

In my opinion, however, proper case selection is far more important than evidence of recurrent sweating It is sufficiently known that limited dorsal sympathectomies (D2–D3) do very well in cases of thromboangiitis obliterans or in simple digital thrombosis, and such cases have been followed by us for 10 to 20 years On the other hand, in Raynaud's syndrome even repeated and more and more extended sympathectomies fail when the phenomenon is an early manifestation of a progressive collagen disease, which per se is certainly unaffected by the operation. I can not remember a single case of Raynaud's syndrome operated on in young women in the absence of any primary cause, which did not turn up years later with scleroderma, with lupus erythematosus, with some hematologic disorder or with some form of compression neuritis which had been overlooked Therefore, the old dictum that one must wait two years before making the diagnosis of "primary Raynaud's disease" by exclusion has to be greatly extended This point is so important that I will quote the brief history of a patient seen several times in our vascular clinic.

> According to my lecture notes, this 36 year old unmarried woman was shown to the students in 1952 as a typical case of Raynaud's disease At about the age of 25, she suffered from excessive coldness of her hands and feet, together with marked perspiration, color changes were definite then in her fingers, but by 1950 her fingertips were ulcerated and scarred A first rib, said to be abnormally high, was removed in a large clinic, without any relief In 1951, bilateral sympathectomies were done at our clinic, which warmed up her hands and healed the ulcers In 1952, when shown to the students, the patient had Raynaud's phenomena and hyperhidrosis in both lower extremities In spite of her long history and because we have been alerted by many previous instances to watch out for scleroderma, there was still not the slightest indication of any such disease Bilateral lumbar sympathectomies of a limited extent were done to prevent excessive perspiration of the trunk
>
> In 1955, 12 years after the obvious onset of her symptoms, the patient returned from her home with a diffuse scleroderma involving the face, hands, arms, feet and lower legs, and an asymptomatic esophageal rigidity on roentgenogram There were few if any vasospastic phenomena in her fingers or toes, but she was badly incapacitated At this writing, she has had two series of treatment with intravenous disodium ethylenediaminetetracetate and has improved, at least temporarily

During the last year, three patients referred for sympathectomy because of Raynaud's syndrome were diagnosed as having collagen disease; one had scleroderma and the other two had false positive Wassermann reactions with high gamma-globulins, obviously early lupus erythematosus The recognition of such a disorder contraindicates operation

Are there any patients then with Raynaud's syndrome who need sympathectomy? Certainly many patients with digital artery thrombosis or stenosis,

with painful ulceration or gangrenous fingertips, who have a *primary vascular disease* with secondary Raynaud's phenomena are greatly benefited by sufficiently extensive dorsal sympathectomies. This is a vast change of attitude, at least in our clinic, since our early indications were directed toward the functional vasospasm with little or no organic lesion. Arteriograms done through the brachial or radial artery are helpful, and the seeming lack of organic obstruction in the presence of clear-cut Raynaud's phenomena is a warning signal of the first order.

2. ACROCYANOSIS

This painless, persistent coldness and cyanosis of the distal parts of the extremities, and occasionally of the lips, tongue, nose and ears, has been recognized for a long time, but its cause remains unknown. On the one hand Lewis and Landis[28] denied the presence of any venous obstruction and spoke about a "local fault," as in Raynaud's syndrome, which maintains heightened arteriolar tone in the arterioles of the skin at ordinary environmental temperatures. It is true enough that the vessels are capable of dilatation brought on by heat or histamine. Day and Klingman[29] found that during sleep the hands of an acrocyanotic patient became warm and red.

On the other hand, according to Villaret and his co-workers,[30] capillary and venous dilatation are the essential lesions in acrocyanosis, based on an endocrine sympathetic failure to maintain adequate tone. They suggest a pituitary hypofunction, but their evidence is meager and indirect. Layani,[31] in a stimulating monograph, marshalled evidence that the venocapillary bed is hyporeactive to epinephrine, that there is a pluriglandular insufficiency and that the venous pressure is constantly elevated.

Richard Capps[32] studied two cases of acrocyanosis with a venous occlusion plethysmograph and noted an abnormal absence of tone of the capillaries, veins and venules. More recently, Edwards[33] described a "remittent necrotizing acrocyanosis," in which, contrary to the previous "functional" forms, there is organic obstruction in the arterioles and capillaries of the skin with perivascular inflammation, hyalin thrombi and endothelial proliferation.

In studying Edwards' 20 cases, one finds that this lesion, not unlike that in Raynaud's syndrome, is truly organic. The remissions of such cyanosis must be due to release of superimposed vasospasm, recanalization or formation of new channels. The sluggish blood flow, perhaps an initial red cell agglutination as reported by Helwig and Freis,[34] may be an important factor.

Here then, again, we have a symptom which needs symptomatic treatment until the etiology is established.

Clinical Picture

The forms our clinic has encountered are characterized as follows:

(1) There is a good history of respiratory infection, perhaps an atypical

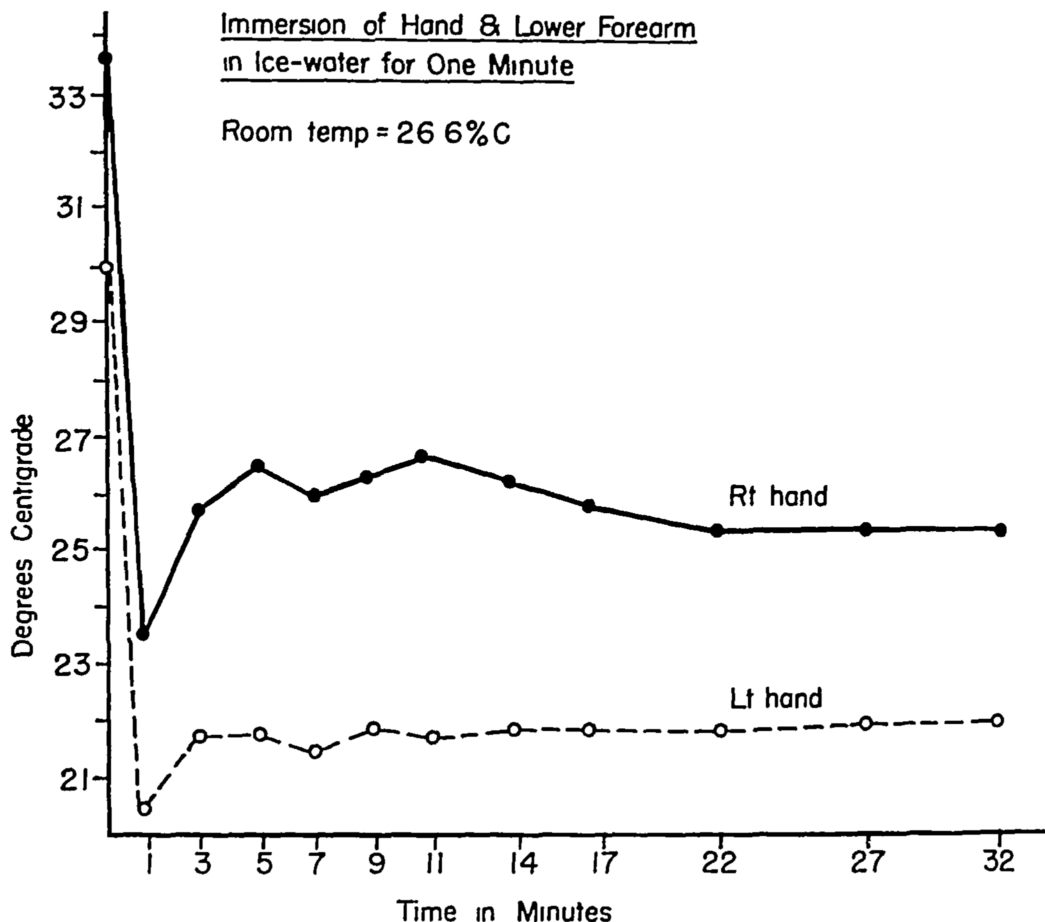


FIG 272 Skin temperatures before and after right dorsal sympathectomy, following immersion of the hand and forearm for one minute, the patient, Mrs R , was suffering from acrocyanosis which was relieved by operation

virus pneumonia, followed by purple discoloration of the hands and possibly other areas on exposure to cold Such patients do show cold hemagglutinins in the serum, a clumping of red cells even on room temperature and a great increase in the sedimentation rate of blood placed in an ice box

(2) The victims are often young to middle-aged women with pluriglandular insufficiency, *i e* , flat sugar-tolerance curve, insulin sensitivity, low basal metabolism and disturbances of menstruation Here, conceivably, a capillary-venous atony is at place Occasionally, patients with Simmonds' pituitary cachexia show acrocyanosis

(3) In the presence of peripheral vascular disease, such as arteriosclerosis with or without diabetes, thromboangiitis obliterans or endarteritis following exposure to cold, fleeting or permanent areas of acrocyanosis develop. These may disappear entirely or become patches of gangrene Capillary and venular thromboses are encountered here with superimposed areas of stasis and sludging, the latter being reversible

These lesions are sometimes accompanied by a livido reticularis, a mottling of the skin over the lower or upper extremities, in which the stasis occurs in the subpapillary venous plexus This livido is aggravated by dependency, by a venous tourniquet and by exposure to cold It is improved by elevation, by sympathetic block or by exposure to heat Histologic studies

in one of our own patients revealed a nodular proliferation in the subpapillary veins,[35] but whether this is the cause or the effect of venous stasis is not clear

Treatment

While heat histamine and paravertebral block rapidly abolish the functional element in acrocyanosis it cannot be said that either these agents or the prolonged use of vasodilators thyroid or ovarian extracts is of any help Nor am I aware of any measure which would influence a positive cold agglutination test or the cold precipitated globulins Sympathectomy however has given excellent results provided (1) The acrocyanosis cannot be a part of a diffuse arterial arteriolar and capillary disease as, for instance in certain diabetic patients In such patients a paradoxical drop in temperature occurs after sympathetic block and terminal circulation is aggravated (2) The skin temperature following sympathectomy must remain *above the critical temperature* at which acrocyanosis occurs (fig. 272) In this respect, acrocyanosis seems to be the mirror image of erythromelalgia where *below a critical temperature* the symptoms of redness burning and throbbing subside (3) When acrocyanosis is associated with diffuse hyperhidrosis which it sometimes is, the anhidrosis following sympathectomy greatly decreases the elimination of heat through that extremity and cyanosis as a result of cooling the extremity is greatly improved

3 ERYTHROMELALGIA

Translated from the Greek this means a red painful extremity but as E. V Allen pointed out,[36] the name does not contain the information that the extremity is warm in fact, that it has to reach a *critical temperature*[37] before the classic symptoms of burning throbbing and tingling begin Anything that will reduce blood flow such as cold wet towels elevation and rest may relieve the symptoms heat, the friction on walking, the covers at night and a dependent position will aggravate them For this reason the term *erythermalgia* was proposed [36] which does introduce the *thermal* element but unfortunately drops the Greek root *erythro* meaning red *erv* in Greek does not convey this and is simply a preposition The original nomenclature coined by Weir Mitchell in 1878[38] is preferable since it is widely used and accepted in the literature

The syndrome has all the earmarks of a symptom and not a disease entity It may be observed in polycythemia and yet in only one extremity and not in the others When the blood count drops the symptoms may improve One observes it in senile arteriosclerotic individuals whose feet are pulseless and expected to be cold yet they are warm tolerate heat or even covers poorly and favor elevation and cooling One may see it in alcoholic polyneuritis, where in one case heat intolerance forced the patient to hang her feet in a bucket of cold water all night, thus adding an immersion limb

(p 149) to her troubles It occurs during tabetic or diabetic neuropathies. We have seen it in an acute lesion of the cord.

One gets the impression that we are dealing with a vasodilator phenomenon, activated by neurogenic stimulation The demyelinization necessary for this process can be readily explained in alcoholic polyneuritis, in tabes, in diabetes and in arteriosclerosis with ischemic peripheral nerves or spinal pathways Just how polycythemia would produce this is unclear, but it is quite possible that the widespread vascular thromboses which occur in polycythemia affect the circulation of the spinal cord or peripheral nerves and produce neurogenic vasodilatation

A most common occurrence is the annoying, intractable burning of the fingers and toes of elderly sclerotic patients, which seems independent of their peripheral vascular sclerosis, sometimes a diagnosis of spinal vascular sclerosis or ischemia of the posterior root ganglia is permissible [39] The anatomic lesion here may be found in the atheromatous occlusion of the spinal arteries at their origin from the aorta. The small, narrow artery coming off at right angles from the spinal artery has been commented on before,[39] and will be discussed with the diabetic neuropathy (p. 445)

The treatment of this symptom can obviously be only symptomatic As previously stated, lowering the blood count in polycythemia alleviates the discomfort A cool whirl-pool bath taken just before bed time may keep the patient comfortable all night, he can install an inexpensive device in his bathtub During the day he gets about in shoes permitting ventilation Radium packs and roentgen ray treatment have been suggested but are of very little help, except indirectly through their effect on the bone marrow Huge doses of thiamin chloride and vitamin B_{12} have been ineffective in our hands Epinephrine by inhalation or injection has been suggested,[40] and in a personally observed case Dexedrine sulfate definitely helped a postural erythromelalgia, heat, throbbing and pain occurred on standing. Sympathetic block or sympathectomy do not often alleviate and may aggravate the symptoms, in a case of erythromelalgia reported in the literature with a diagnosis of thromboangiitis obliterans, a lumbar sympathectomy was performed with no benefit [41] However, in patients with multiple vascular thromboses and a slow stagnant capillary blood flow, sympathetic block should be tried Telford and Simmonds obtained complete relief from the pain of erythromelalgia in three patients subjected to lumbar sympathectomy [42] White and his co-workers had a single example of a brilliant result [21] We have so far not had the courage to do one

4. VASONEUROPATHIES

Vasoneuropathies may be defined as neurovascular lesions which manifest themselves in a combined vascular and neurologic defect, the lesions being caused either by simultaneous action on both vascular and neurologic structures, as in the case of a cervical rib, or by an interaction of these two important peripheral systems on each other While the effect of cold injury

has already been discussed the neurovascular lesions due to compression vibration injuries to the nervous system, lesions of the central nervous system the causalgic state the reflex dystrophy and the diabetic and tabetic neuropathies will be treated more or less as an entity since they have much in common although they exhibit marked clinical differences While the initiation of these lesions by a neurovascular insult may be clear enough the mechanism responsible for the syndrome is debatable

Pathophysiology of the Neurovascular Syndromes

Some authors regard these lesions as being due to irritation of the sympathetic fibers and believe in sympathetic afferents from the extremities analogous to those carrying visceral pain [43, 44] Others postulate sensory sympathetic reflexes, which originate in chronic stimulation of sensory nerve organs around blood vessels, tendinous insertions and joints and which maintain a reflex vasoconstriction and a reflex muscle spasm [45] Finally a hypothesis has been evolved stipulating that the basis of vasomotor phenomena in partial nerve injuries is due to an interaction of fibers within the cross section of a mixed peripheral nerve [46, 47] While in studies from our own group on these interesting painful states we have been greatly influenced by the teachings of Leriche [45] much of the original emphasis on sensory sympathetic spinal reflexes must be modified The present tendency is to evoke short, axon reflexes for many neurovascular phenomena which of course will vary depending on the site of injury A diagram illustrating vasomotor reflexes has been shown in an early communication on post traumatic reflex dystrophy[48] (fig. 273) The afferent arch seems to be carried in the somatic sensory nerves The vasoconstrictor efferents are carried in the anterior roots Whether or not there exists a posterior root efferent vasodilation was debatable then and is still controversial (literature cited in 48) The vasodilator efferent stimuli may go through the sympathetics but can be elicited in the absence of sympathetic outflow in this case they must be axon reflexes or spinal reflexes with an outflow through posterior root efferents These in turn produce vasodilator substances of choline like nature which spread diffusely and defy all patterns of segmental somatic innervation They most likely belong to the parasympathetic, cholinergic system and produce diffuse burning pain Such is the pain in reflex dystrophy in causalgia and in spinal arteriosclerosis This vasodilation can only be abolished when a peripheral nerve is sectioned and time is allowed for degeneration It is significant that causalgic states almost invariably occur in partial nerve injuries, and that when a complete nerve injury is diagnosed this may not be substantiated by histologic section [49]

The idea of Granit and his co-workers[47] that fiber interaction takes place in injured or compressed cross sections of a peripheral nerve has been the most helpful one and men of great experience with causalgia such as J C White[50] and F M Mayfield [49] have subscribed to this explanation

Whether the causalgic state following injury to large peripheral nerve

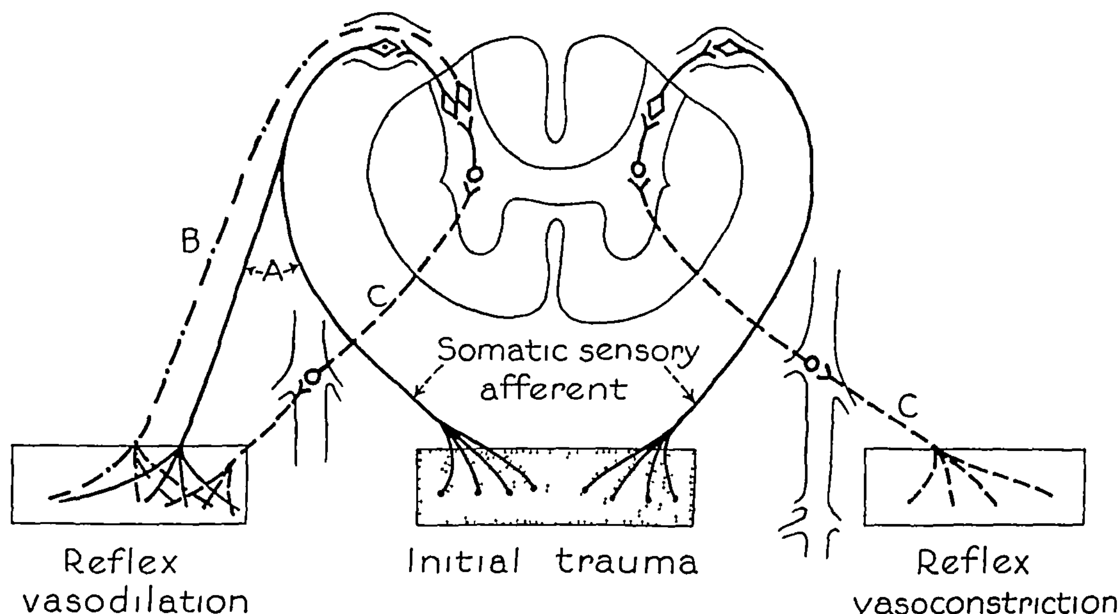


FIG 273 *Trauma* consists of mechanical, chemical, thermal or electric injury, or acute ischemia Somatic sensory fibers carry afferent impulses from the area involved In (A) the impulse never reaches the cord, but produces an efferent vasodilation mediated by posterior root efferent dilators In (B) this same type of vasodilation has been activated through a spinal cord reflex The existence of such a reflex has never been conclusively shown In (C) vasoconstriction occurs through activation of efferent sympathetic fibers, but there are also dilators in this system (Miller and de Takats Posttraumatic Reflex Dystrophy Surg Gynec and Obst, *75* 558, 1942)

trunks, such as the median or sciatic, only differs from the post-traumatic dystrophies by a different site of interaction, *i e*, at sensory nerve endings, is a possibility but is not susceptible to proof

Neurovascular Phenomena Due to Compression or Stretch

Thoracic Outlet Syndrome

There are numerous clinical states in which nerve roots, posterior root ganglia or peripheral nerves are irritated, compressed or stretched. For the upper extremity, the term *thoracic outlet syndrome* has been coined; this really means a neurovascular injury at the level where the brachial plexus, the subclavian artery and subclavian vein cross the isthmus of the root of the neck A number of structures, such as a cervical rib, a high first rib, the scalenus anticus and medius muscles, the pectoralis minor, subclavius and subscapularis muscles and the costocoracoid ligament, and syndromes due to costoclavicular compression and hyperadduction have been described as causing numbness, tingling, vasospasm and even ulceration and gangrene of the fingertips

Cervical rib occurs uncommonly, in about 1 per cent of cases, in most

of these the lesion is asymptomatic or while the anomaly is bilateral, only one side may exhibit symptoms In studying our case histories it also becomes obvious that the patient may go through an asymptomatic period of 20 to 40 years before the presence of the rib becomes noticeable Of the factors which seem to initiate symptoms trauma diminished muscle tone menopause and stooping posture may be mentioned

Even in the absence of roentgen ray evidence of a supernumerary rib there may be a large seventh transverse process and a fibrous or cartilaginous connection between it and the first rib Placing the film in front of a 500 watt light bulb available in roentgen ray departments for purposes of transillumination may visualize such a faint shadow Unfortunately, reproductions of such films are unsatisfactory The compression of the vascular and nervous structures as first stressed by Adson and Coffey [51] occurs between the scalenus anticus muscle and the anomalous rib They recommended an anterior approach a sectioning of the scalenus anticus muscle and if necessary the removal of the protruding part of the first rib Prior to their report, a midcervical or posterior approach had been used to resect a cervical rib and this often resulted in traction on the brachial plexus with anesthesia and paralysis

The center of interest thus shifted to the *scalenus anticus syndrome* Ochsner Gage and DeBakey[52] emphasized that this Naffziger syndrome[53] could occur when the scalenus muscle was hypertrophic or spastic and when the neurovascular structures were unfavorably placed Compression thus

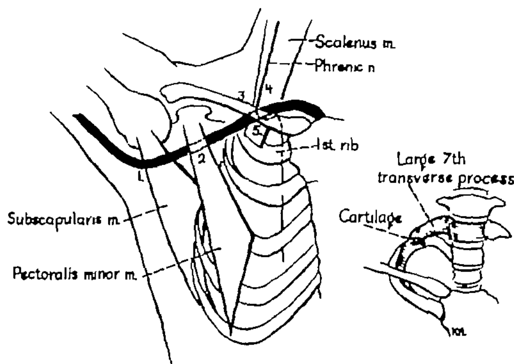


FIG 274 The structures held responsible for the thoracic outlet syndrome are (1) the subscapularis muscle, (2) the pectoralis minor (3) the clavicle, (4) the phrenic nerve and scalenus muscle, (5) the clavicle and first rib. In the insert a cartilaginous connection between the seventh transverse process and first rib is shown. (Redrawn from Hughes, E. S R. Venous Obstruction in the Upper Extremities. Brit. J Surg., *36* 155 1948.)

would be produced against the first rib without the presence of a cervical rib, and many surgeons felt that section or excision of this muscle was all that was necessary to relieve the compression, even in the presence of a cervical rib In addition, the section of this muscle, by allowing the first rib to drop away from the clavicle, increases the space between the clavicle and first rib, thus it counteracts *costoclavicular compression* which, according to Wright and Beyer,[54] occurs during the position of hyperabduction, the arms being brought together above the head with the elbows flexed, a position in which some people sleep

Other structures may compress the neurovascular bundle during certain movements of the arm or shoulder, and Telford and Mottershead[55] thought it highly unlikely that the obliteration of radial pulse could be ascribed to a single cause. Jere Lord, Jr , has removed the clavicle to overcome the costoclavicular compression[56] and more recently has suggested the combination of anterior scalenotomy with the section of the pectoralis minor tendon,[57] another structure which may cause compression during hyperabduction

All of these structures, and in addition the prevenous phrenic nerve, implicated as a cause of subclavian vein compression by Hughes,[58] are depicted in a diagram (fig 274)

Differential Diagnosis

The differential diagnosis of neurovascular syndromes of the shoulder girdle is not always easy [59] The *cervical rib* is, of course, a congenital anomaly, symptoms occurring in early middle life or later These are paresthesias, numbness and pain in the fingers and hands The position which aggravates symptoms or obliterates the radial pulse occurs in adduction of the arm, lifting of weight, as in the case of a red-cap carrying a heavy suitcase, has produced radiation into the arm but also into the anterior chest wall through radiation of pain along the supraclavicular nerves, a form of pseudoangina With the arm in extreme adduction and the head turned to the opposite side, the radial pulse may be obliterated There is roentgenographic evidence of the rib, although the fibrous bands can only be visualized on exploration

The *scalenus anticus syndrome* gives an identical picture There is, of course, no supernumerary rib on roentgenogram and the scalenus muscle is tender on pressure. Infiltrating the muscle with procaine gives temporary relief of symptoms [52] Relief, just as in the case of cervical ribs, may be obtained by hyperabduction and by heat massage or diathermy to the muscles supporting the shoulder girdle, which are often in protective spasm

The *costoclavicular syndrome*, described by Falconer and Wedell,[60] produces a brachial plexus neuralgia identical with the two previous entities, perhaps with more tendency toward Raynaud's phenomena Arterial thrombosis and gangrene have been known to occur The aggravating position is a back and down bracing of the shoulders and hyperextension of the neck, this maneuver, however, closes the pulses in 60 to 70 per cent of normal in-

dividuals Contrary to the scalenus anticus syndrome with or without cervical rib costoclavicular compression causes venous obstruction in some cases demonstrable by phlebogram

The *hyperabduction syndrome* of Wright[61] reveals pallor on hyperabduction but not in all cases The aggravating position is hyperabduction of the arms but as Wright himself has shown [59] the obliteration of the radial pulse occurs in 85 to 90 per cent of normal individuals Thus it is the repeated or prolonged pressure on the neurovascular bundle which causes damage Venous obstruction may occur in some cases The position of relief is extreme adduction The two potential sites of compression are the retroclavicular space between the clavicle and first rib and beneath the tendon of the pectoralis minor muscle

Ruptured nucleus pulposus[62] of the cervical spine produces intermittent paresthesias numbness and pain in the fingers and hand, together with possible roentgen ray evidence of a narrow intervertebral space The aggravating position is extension of the neck, with sudden pressure exerted on the head from above Coughing and sneezing may bring on root pain The myelogram is diagnostic *There is never any evidence of arterial or venous compression* but vasospasm, as in all irritative lesions of the anterior root, may be present Interestingly enough a slipped cervical disk may produce reflex muscle spasm in the neck including that of the scalenus muscle which adds to the diagnostic difficulties This pain usually radiates to the shoulder and exhibits radial distribution so that pain in the index finger aggravated by motion or compression of the cervical spine arouses suspicion I have seen such patients subjected to scalenotomy

The frequency of *cervical spondylosis* or cervical osteoarthritis, in causing numbness tingling and pain in the hands and arms is high In fact, it may cause such compression of the cord and of the anterior and posterior roots including myelographic evidence of pressure by spondylotic ridges that surgical relief by laminectomy by division of the dentate ligament or by actual removal of the offending spur may have to be considered in carefully selected cases [64] Significantly with the exacerbation of symptoms the affected hand gets cooler and may perspire more an irritative lesion of the anterior root carrying the sympathetic efferents

Degenerative lesions of the cord such as syringomyelia and amyotrophic lateral sclerosis, have given rise to an erroneous diagnosis of scalenus syndrome one sees patients who have a full blown neurological lesion with supraclavicular scars as a result of scalenotomy As will be pointed out, scalenotomy has been done many times with insufficient evidence of scalenus compression, or when the muscle spasm is simply a secondary phenomenon Certainly a detailed neurologic examination is in order in every case of the thoracic outlet syndrome

Pleural adhesions at the apex of the lung and the *superior sulcus syndrome* with infiltration of the vertebral foramina may give vasoconstrictor or vasodilator phenomena Roentgenograms taken for osteoarthritis of the spine and for cervical ribs should also include the apex of the lung. An early causalgic

state drew my attention to this area, and an infiltrating lesion was verified by exploration of the chest The case was inoperable

Treatment

Treatment of the thoracic outlet syndrome depends on the accurate localization of compression or stretch of the neurovascular elements. A cervical rib, at least its protruding segment, can be readily removed at anterior scalenotomy, but the presence of a cervical rib does not necessarily mean that it is responsible for all the symptoms I have seen a causalgic state, syringomyelia and amyotrophic lateral sclerosis causing the complaints in the presence of an innocent cervical rib, which remains asymptomatic in roughly half of the patients. Anterior scalenotomy with and without rib resection has been very useful in our hands Distal to the rib, aneurysms and thrombosis of the subclavian artery occur, which is all the more reason for removing the rib in the presence of even mild symptoms [65]

On the other hand, anterior scalenotomy alone and without the presence of a fibrous band often results in failure because (1) the muscle has not been completely divided to include the posterior sheath, (2) the scalenus medius contributes to the compression of the plexus and the artery and must be included in the division, (3) the muscle ends reunite in a fibrous band, for which reason a resection of the muscle belly is advisable, or, (4) most important, the symptoms are not due to scalenus compression and hence the operation was never indicated In our institution the operation is done very seldom and after much hesitation and soul-searching, certainly if a rib is also present it is important to resect it too Otherwise, a second operation may become necessary

There is some experience with the removal of the clavicle for certain patients with costoclavicular compression [66] This drastic procedure should be done only if the minor offending structures have been previously divided without the expected results On the other hand, pectoralis minor division, especially when done in conjunction with aspirating a recent axillary venous thrombosis, is a simple, logical procedure [57]

As a general rule, however, these shoulder girdle syndromes, especially if they produce neuralgic and not vasomotor phenomena, are really managed by the orthopedic and neurologic departments While to patients all numbness and tingling is circulatory disease, to our orthopedic confreres it only becomes so after their procedures have failed

Compression of the Lumbosacral Outflow

While vascular compression at the lumbosacral region is much less frequent than at the thoracic outlet, it may occur, especially at the level of the aorta above the iliac bifurcation where sharp angulations, spurs and kinks do impinge on the posterior wall of the vessel and may help to localize atheromata Compression or traction of the nerve roots, however, due to arth-

rosis to ruptured nucleus pulposus and to the reflex vasomotor effects of muscular and tendinous trigger zones (so thoroughly elucidated by Steindler[67] and by Livingston[68]) produces radiation of pain to the lower extremities which is often ignored and misinterpreted especially when accompanied by vasconstriction or vasodilation

Generally speaking, the neuralgia of a peripheral nerve especially that of the sciatic or femoral and the pain of the lumbosacral disk are accom panied by a decrease in skin temperature What produces an occasional vasodilator response as in an early causalgic state is not readily apparent In diabetic and tabetic neuropathies and in spinal arteriosclerosis this is more apt to be the case as will be pointed out below

Possibly the origin of stimulation is proximal to the posterior root ganglion so that the posterior root efferent fibers are intact or the vasodilators only come into play when the antagonistic sympathetic vasocon strictors are blocked or eliminated In an alcoholic polyneuritis, painful vasodilation was so severe that the patient kept her feet in a cold bucket to obtain relief—a true causalgic state The mechanism of this painful posterior root fiber vasodilation will be further discussed under causalgia (p 434) A paravertebral alcohol block performed elsewhere has led to such a persistent painful vasodilation in the presence of arteriosclerotic ischemia

VASONEUROPATHIES DUE TO VIBRATION (PNEUMATIC HAMMER DISEASE, AIR HAMMER DISEASE)

Pneumatic tools vibrating at a rate from 2,000 to 30 000 times a minute cause numbness, tingling and mostly Raynaud s phenomena There has been some question as to whether the rapid vibration or the percussion is that which sensitizes the terminal nervous and vascular elements of the digit Even a single percussion may lead to long lasting neurovascular phenomena in my experience

Experimentally Denny Brown and Brenner[69] found that percussion of a nerve trunk results in damage to the myelin sheath rupture of Schwann s sheath and escape of damaged myelin into the endoneural space The axis cylinders even of the finest medullated and of the nonmedullated fibers, are not interrupted and do not degenerate However pseudoneuromas develop at the site of injury with edema and the appearance of histiocytes and opportunity for cross stimulation of sympathetic and somatic fibers[46 47] may well occur

In experience gained as a temporary consultant to a large plant in which 30 per cent of the workers using a pneumatic hammer developed Raynaud s syndrome the following factors seem worthy of note the tool itself may be cold or emit a cold exhaust in right handed men the fingers of the left hand exposed to the vibration of the tip in guiding the instrument are most fre quently involved but only at the lower vibratory rates it takes about six months for the symptoms to develop and even although this type of work is promptly stopped the neurovascular phenomena remain stationary for many

years, reassurance does more than any kind of medical or surgical ther including sympathectomy, trophic changes do not develop and the naud's syndrome remains unilateral.

It should be emphasized that this condition may be encountered only in connection with the use of air hammers, but also with chisels, ri road drills and pounding and blasting machines

Biopsies of the skin and subcutaneous tissue of digits affected by hammer disease have been studied by Gurdjian and Walker,[70] these negative On the other hand, Peters,[71] in studying the neurovascular st of workers using a tool vibrating 25,000 times a minute, noted not diminished and absent sensation to touch, pain and temperature in that tion of the hand in closest contact with the tool but also found changes in capillaries of the nail bed in 17 out of 29 patients Instead of the nor hairpin-like loops there was a decided decrease in the affected extren with small, almost occluded vessels in which the corpuscular flow was s gish or absent Neither heat nor cold affected the number or the forn these loops. It is not clear whether this was an organic or functional les

As pointed out in a discussion before the Section of Surgery of American Medical Association,[72] the high vibration rate of these tools f 12,000 to 25,000 times a minute creates a causalgic-like state, with cont ous, burning pain, atrophic musculature, contracted tendons, stiff joints osteoporosis, and represents a percussion neuritis. Patients with high va lar tone or with vulnerable myelin sheaths, such as alcoholic or diat patients, seem to have the most severe air hammer hand

Interestingly, Barker and Hines[73] demonstrated occlusion of branches of the ulnar arteries in patients who complained of "white finge after occupational disease

Since treatment is so unsatisfactory, attempts at prophylaxis are wo while These measures should include (1) limitation of the vibration ur the critical level of 2,000 revolutions per minute, (2) dampening the vibrat by developing a suitable tool, (3) limitation of the length of use of the to and (4) mechanical devices to hold the tool [70,71,72]

I am not sure how many of these recommendations have been put i effect by the industries and how feasible they are The most careful study such patients was reported by Dart from the Medical Department of Chrysler Corporation,[74] who emphasized the presence of high vascular tc either as a predisposing factor or actually produced by the vibration. Sym thectomy has not been helpful in this condition, which is perhaps best ch acterized as a discharging lesion[75] in which a stream of impulses origin from an injured nerve

Vasoneuropathies Due to Injuries of the Nervous System

Since the cross section of peripheral nerves contains somatic and au nomic fibers, a partial injury may selectively inhibit or block certain fib

and leave others intact. It is sufficiently known that direct pressure tourniquet, traction and percussion[69] are demyelinizing factors, and so are cold, diabetes tabes and syringomyelia That the denervated limb or digit is cool is common knowledge most of this is due to the reduced metabolism of paretic muscle although some of it is the result of increased vasoconstrictor tone if the sympathetic fibers are intact [76] While the older literature contains many references to trophic nerves the investigations of Lewis and Pickering[77] led them to conclude that immobility disuse and interruption of motor sensory and autonomic nerve fibers fully explain the vascular phenomena and that there is no reason to postulate the existence of a separate set of trophic fibers Long disuse of a hand leads to a drop in skin temperature but this can be relieved when general cutaneous vasodilation occurs The skin of the denervated cold area will gradually become cyanotic the natural wrinkles will disappear and the skin will become smooth The fingers are tapered their pulp decreases and the nails curve both lengthwise and laterally Since these vascular changes are prominently displayed in causalgia, as in occlusive vascular disease it is well to point out that much of this is simply due to disuse [78] If the patient can begin to use his extremity again either because the pain has been relieved or because his somatic nerves have regenerated these symptoms improve

The vasomotor effects of nerve division have been thoroughly investigated by Richards [79] Immediately after division of a peripheral nerve there is a period of vasomotor paralysis the skin being hot flushed and dry After a variable period usually about three weeks the vasodilation disappears The anesthetic skin is cyanotic and usually colder than the adjacent normally innervated area and trophic changes appear such as digital atrophy loss of skin creases alteration in the growth of the nails and indolent ulcers

The skin of the denervated area loses its independence from environmental temperatures seen in the early phase In this cold phase when the hand is cool the denervated area is cooler when the hand is warm the insensitive digits are warmer Vasomotor reflexes elicited by heat or cold from other normally innervated areas do not affect such digits The caliber of denervated vessels however can be influenced by reactive hyperemia and by tissue metabolites such as accumulate in inflammation

It is of practical importance that axon reflexes such as those elicited by intradermal histamine wheels, cannot be elicited when peripheral nerves have degenerated but are present under spinal anesthesia Our clinic has made widespread use of the histamine response of the skin to test cutaneous circulation and innervation [80] As has been pointed out in part II Methods of Diagnosis (p 58), the absence or presence of this axon reflex can help to map out peripheral nerve lesions and can even help recognize lesions of rapidly conducting myelinated fibers with intact, slowly conducting nonmyelinated fibers The peculiar vasomotor phenomena presenting themselves in such instances will be discussed with the diabetic and tabetic neuropathies (p 445) and with the causalgic states (p 434)

After incomplete lesions of a peripheral nerve one might have. (1) axons that have been completely interrupted, (2) axons that have maintained their anatomic continuity but have impaired conduction, (3) normal axons, and (4) regenerating axons There is general agreement that the small unmyelinated fibers, to which group the vasomotors belong, are the least susceptible to the effects of pressure, traction and percussion, but are more vulnerable to cold and local anesthetics [79] In cases of polyneuritis, a decrease in vasoconstrictor tone is the predominant vasomotor disorder

Abnormal discharge of impulses may occur both in afferent and efferent direction Centripetal impulses produce lancinating attacks of pain; centrifugal impulses passing along vasodilator fibers cause vasodilation and burning

Recovery of reflex vasomotor activity in a denervated area does not necessarily result in a return of normal warmth, this must await the regeneration of fibers responsible for axonal vasodilation [79] Recovery of autonomic function after peripheral nerve injuries suffered in World War II was studied by loss or regain of sweating measured by skin resistance [82] Selverstone and White pointed out that mediation of autonomic impulses may permit widespread activity by a few residual or regenerating axons [82]

Vasoneuropathies Due to Lesions of the Central Nervous System

Anterior Poliomyelitis

During the course of years, mostly through the courtesy of Dr E L Ryerson, I had the opportunity to study the vasomotor status of poliomyelitic limbs It is customary to regard the cold, mottled poliomyelitic extremities as suffering from simple disuse and lack of sufficient muscular activity Investigation of basal blood flow in poliomyelitic limbs found that it was not reduced [83] Nevertheless, in a well defined group of cases the skin is plum-colored and patches of chilblains develop, together with supramalleolar indurations which may break down and show a picture not unlike that of a postphlebitic induration with fat necrosis

The reaction of the paralyzed limb to cold is exaggerated Whether the increased vasoconstrictor tone originates in an involvement of the intermediolateral tract or in demyelinated anterior roots, we are unable to state It is certain that the virus affects other areas than the anterior horn cells

A total of 25 children and adolescents were examined by palpation of pulses, surface temperatures, oscillometric curves and posterior tibial block A typical chart of one case is shown in figure 275 In this unpublished series, nine patients were found in whom a marked vasospasm was demonstrable and, broadly speaking, the extent of the motor paralysis and that of the vascular spasm ran roughly parallel to each other, although some definite exceptions were noted No acceleration of growth was noted after sympathectomy Lumbar sympathectomy with or without excision of such supra-

malleolar patches of cyanosis is conspicuously successful, it helps to heal pressure sores from braces or incisions from orthopedic procedures done for stabilization of the ankle or muscle transplant (fig 275) Following sympathectomy the limb remains warm the ulcer heals and many of the nodular thickenings disappear

C se No 9955
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NEUROCIRCULATORY CLINIC

N me E. Z Ag 19 Diagnosis Ant. pol omyelitis trophic ulc of ankl

Pul e

	Right	Left
F mo al		
Popl teal	3	3
Post tib al	3	1
Do sali pedis	2	1

H tam n t st Right L ft
On the ba of 0 to 3

	Right	Left
Above knee	3	3
B low kn	3	2
M ddl of calf		
Ankl	2	1

Blood p e sur
Blood volume
Blood count
Blood vi cosity

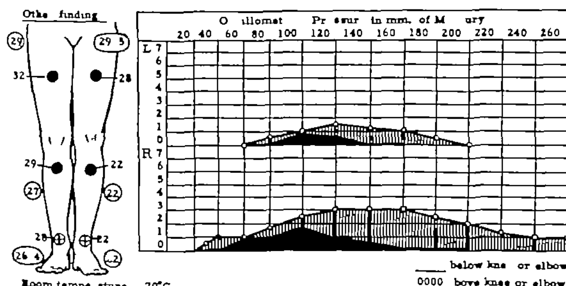


FIG 275 The neurocirculatory record of E. Z., a 19 year old girl, suffering from a poliomyelitic left lower limb, with a trophic ulcer and marked cold sensitivity Full circles represent normal histamine flares shaded or crossed circles are diminished flares Note the decreased skin temperatures, the diminished oscillometric curves both above and below the knee and some but not full increase in skin temperatures after posterior tibial block. This was the first patient sympathectomized for poliomyelitis in our clinic. (Unpublished observations of E. L. Ryerson and G de Takats.)



FIG 276 This 40 year old man was referred to us as a case of Buerger's disease Multiple spontaneous painless amputations of fingers have taken place The toes are also involved

Progressive Muscular Atrophy

The hand and lower arm in progressive muscular atrophy are cool and often cyanotic This, as in anterior poliomyelitis, is partly due to disuse and partly due to the involvement of vasomotor centers in the gray matter of the cord and involvement of the vasomotor outflow in the anterior roots Outside of testing for faradic and galvanic irritability and for the reaction to degeneration, the typical fibrillation of muscles can be looked for, especially when the muscles are put on slight stretch or tapped I saw a young doctor operated upon in another institution for scalenus syndrome, who on examination showed unequivocal evidence of progressive muscular atrophy At any rate, an orderly neurologic examination should be performed, since in the early stages of these muscular dystrophies the diagnosis of vascular disease may be made and a number of unwarranted operations be performed The classification and separation of these myopathies can be studied in neurologic texts [84]

Syringomyelia

Because the gliosis or cavity formation compresses or irritates the anterior horn cells and involves the lateral horns of the gray substance, vasomotor disturbances in the parts corresponding to the muscular atrophy, bone atrophy and sensory changes are frequent [84] The hands are moist and bathed in cold perspiration, they are edematous and deeply cyanotic, which is ac-

centuated in the dependent position This *cheiromegaly* has been termed main succulante by the French and in vascular clinics such cyanotic hands and feet together with Raynaud s syndrome hypothenar atrophy and a sensory loss of the first dorsal and eighth cervical segments, suggest a cervical rib To complicate things a cervical rib may be present in cases of syringomyelia [85]

The trophic changes include thickened skin painless whitlows indolent ulcers developing from a cigarette burn and other injuries all of which are painless *Morvan s disease* is a form of syringomyelia in which all sensory fibers serving a certain area are destroyed leading to spectacular patches of ulceration or gangrene (fig 276) Cases of familial lumbosacral syringomyelia with conspicuous trophic disturbances including gangrene are on record

The surgeon while not frequently may be tempted to regard these patients as suitable for sympathectomy His role in the vascular clinics is to ferret out these neurologic cases and protect them from operation

Hemiplegia

Shortly after the cerebrovascular accident, the affected extremity is warmer and somewhat puffy and looks as if sympathetic pathways had been transected Later the paralyzed extremity becomes cool, the fingers tapered and the muscle atrophic as in other forms of disuse There is however a rare occurrence of a severe burning pain the so-called thalamic pain which has a causalgia like character This contralateral dysesthesia and pain has been known to continue unabated even as long as eight years [87]

Neurosurgical methods have been authoritatively and exhaustively discussed by White and Sweet in their monograph on pain [50] Here I just wish to register some observations made on these painful hemiplegias with para vertebral sympathetic blocks

If one blocks the affected side the edema may temporarily disappear but the pain is uninfluenced In two instances, the cervical sympathetic chain was blocked contralateral to the hemiplegic upper extremity on the side of the cerebral lesion in both cases marked relief occurred one lasting for weeks and in the other for several months Since according to Walker [88] the thalamus receives its blood supply from five sources any one of these arteries, and most importantly the thalamogeniculate artery may be blocked the resulting collateral edema can be influenced by sympathetic paralysis such as our clinic has employed it in the emergency treatment of apoplexy [89] This practical approach should be attempted early before the organic lesion becomes irreversibly fixed It simply influences the halo of edema around the infarct As will be discussed under causalgia (p 434) this central thalamic pain represents lesions of the third sensory neuron, and is seen after trauma to the cortex, after certain tumors and after cortical infarcts In my own limited experience such patients often commit suicide, end up in institutions or become addicted to narcotics [90]

The Causalgic State

Although this syndrome had been previously recognized, its description by Mitchell, Morehouse and Keen in 1864, based on observations of soldiers wounded in the Civil War, is classic.[91] Large wars have always contributed to the understanding of this "painful, long-continued, unendurable pain and its influence on body and mind."[92] Thus, based mostly on his experience in World War I casualties, Leriche[93] pointed out the advantages of early periarterial stripping and arteriectomy, and later gave a searching analysis of the symptoms, believing them to be the result of wounded sympathetics.[94] Following World War II, the studies of White, Mayfield and Rasmussen were particularly impressive (literature cited in 49). Because of some experience with civilian injuries and the interest of our group in the so-called reflex dystrophies, it was felt that the minor causalgias, named by John Homans,[95] and the true causalgias differ only in the site of the injury and in the degree of pain, but are not necessarily separate clinical entities. Arguments for this concept, which by no means is acceptable to all authors, will be briefly outlined in discussing their common and their divergent features.

Symptoms and Course

The *onset* of severe, excruciating, burning pain is immediate in most major and minor causalgias. There is an occasional case in which the onset of pain is delayed for 24 to 48 hours after the injury, and very rarely the symptoms do not appear for two or three weeks. In the major causalgias, the median or sciatic nerves are involved most frequently; in the minor ones, which we have described under post-traumatic dystrophy of the extremities,[96] the radial interosseus, the saphenous superficial peroneal and the posterior tibial nerves have been overstretched or bruised, but in not a single one of our patients has there been a complete nerve injury. This would support the hypothesis that intact sympathetics stimulate demyelinated sensory fibers which, in turn, conduct the stimulus to the periphery, causing the secretion of vasodilator substances and centrally producing pain.

The nature of this painful vasodilation was discussed from our clinic in 1943.[97] Everyone is familiar with the vasodilation as a result of direct heat, intra-arterial papaverine, peripheral nerve block, paravertebral sympathetic block, nitrites and the ganglioplegic agents such as hexamethonium or tetraethylammonium chloride. But the vasodilation associated with the minor and major causalgias must have a different mechanism, since sympathectomy does not abolish it but may even increase it, even though the pain is relieved. The vasodilation can be increased by a parasympathetic stimulant such as neostigmine (Prostigmin), Mecholyl or Urecholine, whether the sympathetics are present or not. Neostigmine has been used to accentuate the painful vasodilator state after injuries, and such a situation is shown in figure 277. Pain and edema were aggravated on the affected side after the

injection of neostigmine and the oscillometric index rose The vasodilation on the other hand together with its painful throbbing character is abolished by moist cold by arterial compression and by keeping the extremity absolutely quiet eliminating all external stimuli such as noises jarring or drafts

The thought was expressed in 1943[97] that the presence of capillary hypertension would readily explain all these observations The explanation of a *spreading neuralgia* occurring both in the major and minor causalgias, a neuralgia which spreads in a central direction from the injured periphery runs into difficulty It is pertinent to the case in point that the peripheral extensions of the autonomic nervous system do not undergo degeneration when the related postganglionic fibers are divided [98a] This terminal nerve network which can be stained by vital methylene blue is formed by autonomic interstitial ganglion cells The nerve impulses or the hormonal and other humeral changes in the environment of the interstitial cells lead to the production or liberation of chemical mediators which spread in decremental fashion through the network Depending on the intensity of the impulses the spread may be relatively limited or widespread with consequent variations in the motor response [98b]

Neither in the symptomatology nor in the progression of pain edema spreading neuralgia and the consequent emotional changes, does there seem to be any fundamental difference between the major causalgia following partial injury of a large mixed peripheral nerve and the minor causalgia, which occurs after comparatively mild blunt injuries and which Miller and I studied in its different stages and different rates of progression [96]

Ever since Sudeck described puzzling vasomotor phenomena following comparatively mild injuries occurring mainly at or below the wrist and ankle [99] a variety of terms have been employed to characterize the same syndrome *i e* traumatic angiospasm, chronic traumatic edema acute atrophy of bone post traumatic osteoporosis and reflex nervous dystrophy

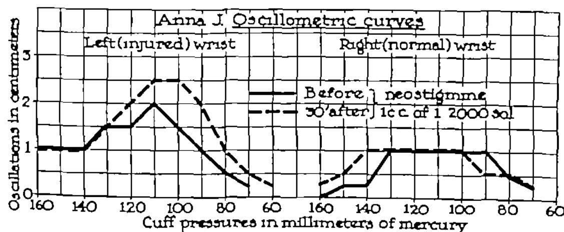


FIG 277 Oscillometric curves in a patient who suffered from a painful wrist with spreading neuralgia and increased oscillations following a streetcar accident. Note that the oscillations are higher on the injured side and are markedly exaggerated after the use of neostigmine. In the right, normal wrist neostigmine did not increase the oscillations, although a slight shift to the left (increased peripheral resistance) took place. (de Takats, G Nature of Painful Vasodilatation in Causalgic States. Arch Neurol and Psychiat., 50 318 1943)

We pointed out later that rheumatic arthritis with its vascular phenomenon, the frozen shoulder after myocardial infarction and the major causalgias have an identical three-stage course, although they admittedly differ in intensity and prognosis

In studying the early phenomena after comparatively mild trauma, one can establish a first stage of severe, persistent pain with a burning character aggravated by jarring, air currents, noises and emotional upsets If the injured limb is properly immobilized, uninfected and seemingly on the way to normal repair, such complaints should make one suspicious of an early causalgic state At this stage, the pain is closely limited to the site of injury, the extremity is warm and dry but edematous and the muscles are in a protective spasm to splint the painful area There is no osteoporosis as yet Studies of blood flow with a water plethysmograph and with oscillometric curves indicate increased circulation to the part,[100] which, of course, is partly metabolic, being the response of the tissues to injury and mediated by tissue products Anyone's mashed finger is hot and throbbing, *but why doesn't it subside?*

It is here where one has to invoke the vasomotor reflexes, which certainly operate after all trauma As early as 1924, Fritz Albert showed experimentally the presence of these reflexes, which were predominantly vasodilator [101] Trauma in the broad sense of the word may consist of mechanical, chemical, thermal or electric injury As shown in figure 273, both axon reflexes and posterior root efferents, whose existence is still debated, can mediate cholinergic vasodilator reflexes This neurohumoral vasodilation can be eliminated by sympathetic block, either because the more rapid flow of blood "washes out" these substances,[102] or, as seems more likely, the choline-like vasodilators are destroyed rapidly in the presence of adequate blood flow Reflex vasoconstriction, which is always present though masked by vasodilation, prevents the destruction of acetylcholine by cholinesterase by creating a shift of pH to the acid side [103] Similarly slight changes in oxygen tension of the tissues which occur in vasoconstriction cause an appreciable inhibition of amine oxidase, the inactivating agent of adrenergic nervous mechanisms [104] This difficult field will no doubt be further studied by suitable methods determining cholinesterase, acetylcholine, amine oxidase and norepinephrine in the tissues Progress will here depend on the activity of histochemists

This vasodilator preponderance gives way in a *second stage* to a cold, perspiring, vasoconstricted extremity with hard, cyanotic, nonpitting edema The joints have become stiff and the muscles are becoming atrophic Osteoporosis, which in our experience does not become apparent for six to eight weeks after the injury, is now definite Many of the phenomena now seen are the result of disuse, but use of the limb is not resumed because of pain and fear of pain. Osteoporosis is now evident, first spotty and later diffuse. Blood flow is still unstable and its reaction is exaggerated to heat and sluggish to cold The pain is not limited any more to the site of injury, but takes on the character of a spreading neuralgia, a hyperalgesia which may or may not

follow segmental distribution Thus a small injury to a digit may end in a hyperalgesia of the elbow shoulder and anterior chest wall not unlike a cutaneous hyperesthesia in visceral disease

Paravertebral sympathetic block which must extend from the second to the fourth dorsal ganglia for the upper extremity and from the first to the fourth lumbar ganglia for the lower extremity will warm the limb abolish or diminish the edema *even without too much active motion* and permit painless active and passive motion The specific action of sympathetic block is diffi cult to explain unless one is willing to assume afferent viscerosensory fibers coursing in the sympathetics The majority of investigators, including J C White [105] doubt that sympathetic axons are of any importance as an accessory pathway for pain originating in the extremities It may well be however as suggested by Cooper and Kerslake [106] that reflex thermoregulation in the skin is mediated by sympathetic fibers and thus modifies the number of im pulses passing up the afferent fibers and triggering the paroxysm of pain

This second stage if adequately treated is still a reversible one, although it responds much more slowly to treatment than the first stage If untreated or mistreated it will progress to the *third stage* Here there is a general atrophy of tissues including skin muscle and bone The extremity is cold and clammy The tendons are shortened the joints are ankylosed The osteoporosis is diffuse and indistinguishable from any other type of osteoporosis Pain is now intractable The patient is completely demoralized He or she is addicted to narcotics may be committed to an asylum or may commit suicide

A few illustrations may serve some of the points just made

In figure 278 a post traumatic dystrophy is shown six months after an unrec ognized fracture of the fibula with ankle sprain in a 55 year old railroad employee. There is localized hypertension in the affected right limb After the paravertebral block, which gave complete relief from pain, the spikes became higher but the previously high diastolic pressure which registered at 70 mm. Hg now drops to 40 mm. Hg. The throbbing, vascular pain relieved by cold towels and aggravated by heat disappeared, and after a few repeated blocks complete restoration of function was obtained.

In a later third stage of this syndrome, the right hand of a 24 year old musician is shown who fell on the ice while skating. While no fracture or dislocation was found there was immediate burning pain from the start. Much splinting, physical therapy and manipulation was done at a large university orthopedic service, but these measures aggravated the patient s symptoms. This hand, seen four years after the injury (fig. 279) was cold and cyanotic. The exten sor muscles were so atrophic that active dorsiflexion was impossible, although the muscles reacted well to electrical stimulation. The hand was so painful that physical therapy could only be given under brachial plexus block. Full recovery as a violinist could not be expected, but psychotherapy was started. She returned to the East and her case could not be followed. Her osteoporosis is shown in figure 280, with severe irreversible joint and tendon damage.

Of the over 100 blood flow determinations made on this type of patient with Donald S Miller [100] only one will be shown to demonstrate increased blood flow in such a limb and its sluggish vasoconstrictor response to cold (fig 281)

Conversely heat will exaggerate the increased blood flow in the affected extremity as shown in figure 282 In some cases such as in case 1 there is

Tycos Recording — Garrity L before block
Tycos Recording — Garrity R before block
Tycos Recording — Garrity L after block
Tycos Recording — Garrity R after block

FIG 278 High oscillometric spikes are shown on the right injured side, due to a partial superficial peroneal nerve injury with an unrecognized fracture of the fibula After paravertebral block there is a demonstrably *increased* flow with *decreased* peripheral resistance Clinically, pain was relieved, edema subsided and pain on weight bearing was abolished



FIG 279 This is the cold, glossy, atrophic right hand of 24 year old Helen L , a professional musician, who fell on ice four years before this picture was taken There is ulnar deviation due to atrophy of ulnar extensors and her thumb (not visible) is subluxated (The "hysterical hand" of the psychiatrist)

FIG 280 Note the severe spotty osteoporosis in the right hand of Helen L. (shown also in fig. 279) Some coarse trabeculation is visible, indicating that recalcification is under way The joint spaces are narrow or obliterated. The atrophy of the ulnar extensors resulted in a deviation of the hand toward the radial side. The thumb shows subluxation, not a result of the original injury

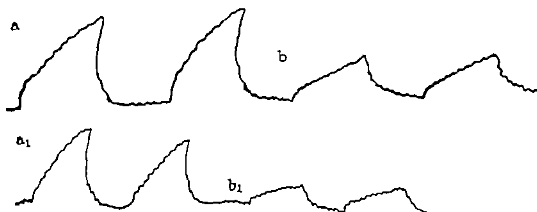


FIG 281 Blood flow in an injured right hand (a) at 32 C. and (b) at 17° C., together with the control flows in the left hand (a_1) and (b_1) The blood flow in the injured hand dropped from 87 cc. of blood per 100 cc. of tissue to 53 cc. of blood per 100 cc. of tissue. For the normal hand the corresponding figures were 75 cc. and 27 cc., respectively All these figures represent averages of five to seven trials.[14]

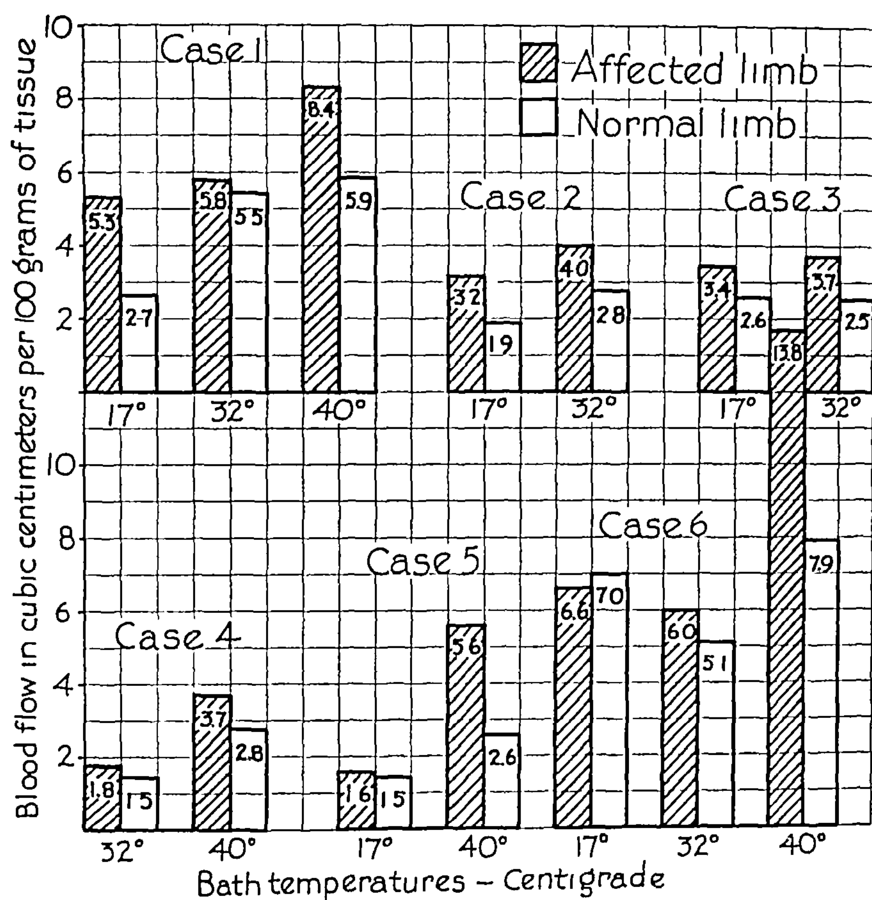


FIG 282 The effect of environmental temperatures on blood flow in Sudeck's atrophy Note that in each instance, the hot water bath raises the blood flow more on the injured side, even if there is little difference in the blood flows in cold and moderate environments This was especially marked in case 6

hardly any difference in flow at normal temperatures (32° C , 88° F.) Both cold and warm environment bring out the difference Note the tremendous vasodilator response to heat in case 6, in which at cold and at normal temperatures there was little difference in flow between the injured and uninjured extremity

There may be several reasons why some authors could not find this vasodilator phenomenon in major causalgic states J C White and his coworkers[107] have pointed out that many of the war injuries were seen comparatively late, when increased sympathetic overactivity with coldness, cyanosis and increased perspiration were prominent features Shumacker and Abramson,[108] together with many other authors including Holden,[109] believe that the post-traumatic vasomotor disorders should be strictly differentiated from causalgic states Mayfield[49] believes that causalgia must be regarded as a clinical entity, though it may arise from a common pain mechanism with certain of the minor causalgias

Since the total material of 54 personally observed cases[90] actually relates to civilian injuries, mostly due to a stretch of peripheral nerves at the wrist and ankle, the question can immediately be raised as to their proper inclusion into causalgic states

The dramatic war injuries affected young, healthy and exceptionally

brave soldiers and turned them into physical and mental wrecks within a few weeks time The injuries were massive due to shell fragments or bullets affecting, *but not completely severing* the brachial plexus the median sciatic and occasionally the tibial or peroneal nerves In Mayfield s carefully tabu lated series of personal cases, cases reported in the literature and cases seen in the U.S Army Neurosurgical Centers the incidence of causalgia in its severe form occurs in 2 to 5 per cent of all major nerve injuries [49] But as pointed out by Echlin 19 6 per cent of the patients with nerve wounds suffer causalgia in some degree but they improve before they reach a neurosurgical unit

Causalgia which I have only seen on occasional visits to vascular centers of army hospitals or as late cases at the Great Lakes Naval Hospital and the Veterans Facility is simply a vastly accelerated form of the post traumatic reflex dystrophy which affects smaller nerves often synovial and tendinous nerve receptors this is then protracted by ill advised physical therapy heat treatment the anxiety of the patient and his occasional cupidity to obtain financial gain from his industrial injury In other words the intensity of the injury the poor recognition and initial treatment in first aid stations, the pressure of claim agents, the activity of ambulance chasers and the age and financial insecurity of the patient add a compensation neurosis to the picture It should be hastily added however that one can see post traumatic dys trophies with none of these elements present and can see it in its early phases without the superimposed anxiety, insecurity and fear all of which hasten the predominance of sympathetic overactivity

Basically then, the causalgic state is due to a partial nerve injury setting up reflex and efferent vasomotor phenomena which at first are predominantly vasodilator Interestingly half of Mayfield s causalgias obtained relief from cold 38 preferring cold applications 30 preferring warm applications and 7 deriving no benefit from hot or cold moisture many obtained temporary relief for the first time in many weeks

The personality change is the effect of severe prolonged suffering, not its cause as many psychiatrists and analysts would lead one to believe It is very true however that in the minor causalgias the patient s unwillingness to move and use a painful extremity greatly contributes to the protraction of the disease bringing on some of the results of disuse and lack of weight bearing, such as coldness cyanosis and stiffness of joints

Thus the essential difference between major and minor causalgias is (1) the intensity of the injury and its accelerated rate of progress (2) the size and sympathetic component in the mixed peripheral nerve, and (3) the early skill ful institutional treatment of these casualties as administered in hospitals of the Armed Forces especially geared for such cases

On the other hand the post traumatic dystrophies (1) follow mild blunt trauma, (2) are frequently misinterpreted and mistreated, and (3) are com plicated by mutual mistrust between patient and industrial physician by protracted litigations, by offers of settlement and by the development of a state of mind

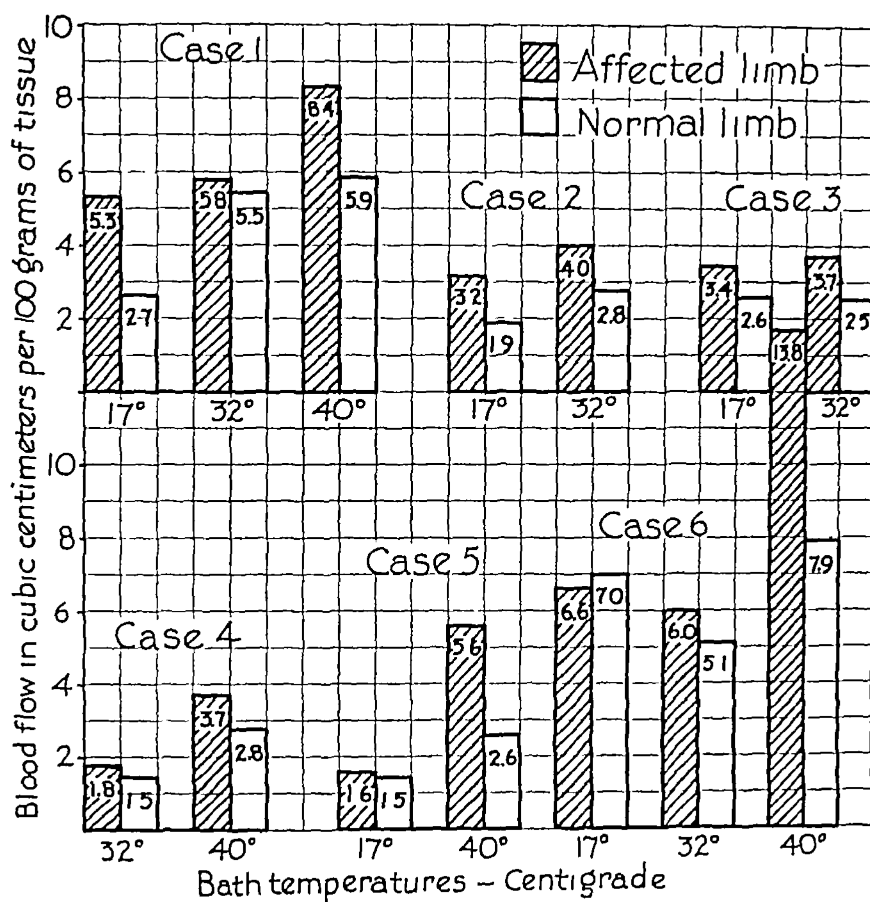


FIG 282 The effect of environmental temperatures on blood flow in Sudeck's atrophy Note that in each instance, the hot water bath raises the blood flow more on the injured side, even if there is little difference in the blood flows in cold and moderate environments This was especially marked in case 6

hardly any difference in flow at normal temperatures (32° C, 88° F) Both cold and warm environment bring out the difference Note the tremendous vasodilator response to heat in case 6, in which at cold and at normal temperatures there was little difference in flow between the injured and uninjured extremity

There may be several reasons why some authors could not find this vasodilator phenomenon in major causalgic states J C White and his coworkers[107] have pointed out that many of the war injuries were seen comparatively late, when increased sympathetic overactivity with coldness, cyanosis and increased perspiration were prominent features Shumacker and Abramson,[108] together with many other authors including Holden,[109] believe that the post-traumatic vasomotor disorders should be strictly differentiated from causalgic states Mayfield[49] believes that causalgia must be regarded as a clinical entity, though it may arise from a common pain mechanism with certain of the minor causalgias

Since the total material of 54 personally observed cases[90] actually relates to civilian injuries, mostly due to a stretch of peripheral nerves at the wrist and ankle, the question can immediately be raised as to their proper inclusion into causalgic states

The dramatic war injuries affected young, healthy and exceptionally

brave soldiers and turned them into physical and mental wrecks within a few weeks time The injuries were massive due to shell fragments or bullets affecting, *but not completely severing* the brachial plexus the median, sciatic and occasionally the tibial or peroneal nerves In Mayfield s carefully tabu lated series of personal cases cases reported in the literature and cases seen in the U S Army Neurosurgical Centers the incidence of causalgia in its severe form occurs in 2 to 5 per cent of all major nerve injuries [49] But as pointed out by Echlin 19 6 per cent of the patients with nerve wounds suffer causalgia in some degree but they improve before they reach a neurosurgical unit

Causalgia which I have only seen on occasional visits to vascular centers of army hospitals or as late cases at the Great Lakes Naval Hospital and the Veterans Facility is simply a vastly accelerated form of the post traumatic reflex dystrophy which affects smaller nerves often synovial and tendinous nerve receptors this is then protracted by ill advised physical therapy heat treatment the anxiety of the patient and his occasional cupidity to obtain financial gain from his industrial injury In other words the intensity of the injury the poor recognition and initial treatment in first aid stations the pressure of claim agents the activity of ambulance chasers and the age and financial insecurity of the patient add a compensation neurosis to the picture It should be hastily added however that one can see post traumatic dys trophies with none of these elements present and can see it in its early phases without the superimposed anxiety, insecurity and fear all of which hasten the predominance of sympathetic overactivity

Basically then, the causalgic state is due to a partial nerve injury setting up reflex and efferent vasomotor phenomena which at first are predominantly vasodilator Interestingly half of Mayfield s causalgias obtained relief from cold 38 preferring cold applications 30 preferring warm applications and 7 deriving no benefit from hot or cold moisture many obtained temporary relief for the first time in many weeks

The personality change is the effect of severe prolonged suffering not its cause as many psychiatrists and analysts would lead one to believe It is very true however that in the minor causalgias the patient s unwillingness to move and use a painful extremity greatly contributes to the protraction of the disease, bringing on some of the results of disuse and lack of weight bearing, such as coldness cyanosis and stiffness of joints

Thus the essential difference between major and minor causalgias is (1) the intensity of the injury and its accelerated rate of progress (2) the size and sympathetic component in the mixed peripheral nerve and (3) the early skill ful institutional treatment of these casualties as administered in hospitals of the Armed Forces especially geared for such cases

On the other hand the post traumatic dystrophies (1) follow mild blunt trauma, (2) are frequently misinterpreted and mistreated and (3) are com plicated by mutual mistrust between patient and industrial physician by protracted litigations by offers of settlement and by the development of a state of mind

The interesting fact that only about 5 per cent of all major nerve injuries exhibit the causalgic state,[49] and that there is 5 per cent of post-traumatic vasospasm among 500 sprains, makes one curious about the personality makeup of persons prone to develop this complication Certainly the war experience has shown that the most stable and the healthiest individuals when receiving a certain type of wound develop causalgia This is not always clear in the civilian industrial or traffic injuries If one accepts the cross stimulation theory of Doupe, Cullen and Chance,[46] one has to postulate that the nerve be always partially injured so that efferent vasodilator conduction is possible, and that sufficient sympathetic activity or possibly hyperactivity be present to activate these vasodilator efferent fibers One line of approach which has, to our knowledge, not been pursued would be to test these patients for central sympathetic reactivity with a simple clinical test,[110] but even here the injury itself and the previous management of the case may modify the reaction

Treatment

Irrespective of whether the burning paroxysms of pain are carried in the sympathetics, a hypothesis which still has adherents,[97] or whether the sympathetic efferents stimulate demyelinated posterior root fibers,[46, 47] the effect of sympathetic block on this painful syndrome is specific This is so true that a failure of a paravertebral block to relieve causalgia either means that the block was faulty or not extensive enough for sympathetic denervation of the affected part, or that the lesion is not a true causalgic state, the pain having spread to higher sensory levels at cord or midbrain

In both the major causalgias and the minor post-traumatic dystrophies, early sympathetic blocks are urgent. For the upper extremity it is well to include the first to fourth dorsal segments, so-called stellate blocks to the cervical sympathetics often give only partial or insufficient relief For the lower extremity, the involvement of the thigh calls for injecting the first lumbar segment, and should the buttocks be the site of pain, the block has to extend to the tenth dorsal

In a true causalgic state, seen within the first few weeks or months following the injury, the block gives relief for 12 to 24 hours, only to recur again Should the effect of block last for weeks one might as well use a sham injection of physiologic saline solution The effect of such a placebo, which I have administered in several suspicious cases, is startling and saves both patient and surgeon from an unnecessary sympathectomy Both hysteria and malingering can be excluded with the help of such a placebo, and both types of patients can be readily identified with maneuvers familiar to neurologists

While a few patients in our series did benefit from repeated sympathetic blocks and, while under the protective umbrella of the painless state, active and passive physical therapy can be started, the more severe cases and certainly those in the second stage require complete surgical sympathectomy Mayfield's emphasis on extending lumbar sympathectomy to the first lumbar

or even to the tenth dorsal segment has been fully confirmed in our clinic, and we have demonstrated to the unbelieving neurosurgical residents that a causalgia following a sciatic nerve injury can only be relieved by a lumbodorsal sympathectomy On the upper extremity a combined axillary neurovascular injury with residual nerve lesion was not relieved until an extended dorsal sympathectomy was performed

There is, of course another source of failure which was pointed out some time ago with the help of a diagram (fig 283) If one looks on the sensory pathways as being susceptible of stimulation at any level as forcibly emphasized by Kinnier Wilson,[111] one can classify causalgic phenomena accordingly into lesions of the first second or third sensory neuron Peripheral trauma activates the first neuron producing vasomotor reflexes which under certain conditions are predominantly vasodilator Contusion concussion or stretch operates in the minor causalgias gunshot wounds of large peripheral nerves operate in the major causalgias Early treatment by sympathectomy will eliminate these painful vasomotor phenomena although the sensory nerve lesion will of course, remain

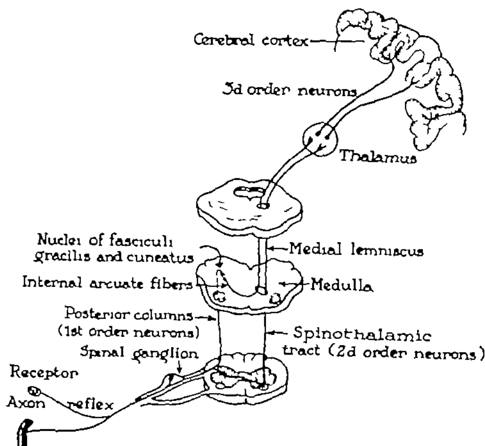


FIG 283 Diagram of the three sensory levels. The peripheral (first) neuron has its ganglion cell in the posterior root ganglion. The peripheral processes of these ganglion cells form the sensory nerves ending in special receptors in skin, muscle or tendon. The central processes pass through the dorsal roots into the spinal column where some of them synapse with second order neurons. While the posterior columns are still first order neurons, the spinothalamic tract constitutes a second order neuron, connecting in the thalamus with the highest third order sensory neuron. (de Takats, G Causalgic States in Peace and War J.A.M.A 128 699 1945)

Recognition of a lesion of the second neuron is not difficult when other localizing signs are present Poliomyelitis is often very painful, when the virus invades the internuncial neurons[112] or demyelinizes the anterior roots, giving opportunity for cross stimulation, muscular hypertonus, reflex hyperirritability, burning pain, heat and edema develop We have seen extremities in patients suffering from tabes, from combined degeneration of the cord or from acute postscarlatinal myelitis showing causalgic states [90] While such lesions are of neurologic interest, their real significance lies in the fact that the first neuron lesion, the early causalgic state, may ascend to this level if unrecognized or mistreated, and may produce the same diffuse intractable dysesthesia with mirror images to contralateral, symmetrical areas Livingston[68] has postulated that the internuncial neurons become hyperirritable in the presence of persistent peripheral impulses, and that a sensory barrage does produce chemical and electrical phenomena in the cord is documented by good evidence [114] Such a cord is then comparable to that of an experimental animal whose cord has been exposed to strychnine

Clinically, in this stage of spreading neuralgia and diffuse hyperesthesia with burning pain, paravertebral blocks still give relief But should sensory phenomena relating to the third highest neuron appear, peripheral block of sympathetics, or as J C. White[107] pointed out, spinal anesthesia or brachial plexus block, will not afford relief These third sensory neuron lesions which occur after cortical trauma, infarction of the sensory cortex or tumors of the cortex produce painful vasomotor phenomena, not unlike causalgia [115] Again, the fact must be kept in mind that the barrage of continuous impulses, originating from lower sensory levels, so stamp their impression on the sensory cortex that it becomes the seat of a central pain, such as one sees in phantom limbs not responding to cordotomy

The whole question of central pain and its surgical management is outside the scope of this monograph, and has been extensively and searchingly treated in White and Sweet's monograph on pain.[50] The only point to be made here is that failure of paravertebral block or of peripheral nerve block to temporarily eliminate causalgic pain means that higher sensory neurons are involved and that sympathectomy is going to be a failure Here again there is a difference between the early major causalgias seen in the neurosurgical and vascular centers of the armed services and the long-protracted civilian injuries, where the original injury is minor but the duration of pain is much longer Pain is a progressive disease and should be blocked as early as possible.

Other forms of therapy outside of sympathectomy should be mentioned Echlin and his co-workers[115] made the point that many causalgias spontaneously recover or are ameliorated before they reach a surgical center, while he estimated that an incidence of 19 6 per cent of causalgias occur sometime after peripheral nerve injury, he only found 2 per cent of the cases severe enough to require sympathectomy when they reached his center Again, among the civilian injuries many patients treated by immobilization alternating with physical therapy, local injections of procaine and hydro-

cortisone and ganglioplegic agents such as hexamethonium or tetraethyl ammonium chloride may gradually and slowly regain function and lose their pain over a period of many months or years

There can be no objection to a trial of such therapy if it results in definite improvement It may well be that in cases of patchy demyelinization the fragmented myelin sheath can regenerate [69] No time should be lost, however if the causalgic state persists or progresses for fear that it will ascend to higher sensory neurons In such a case sympathectomy is of no avail nor have sensory tract sections in the cord or brain given dependable and permanent relief [50]

Resection of the partially damaged nerve with resuture may help a causalgic state but may of course result in a larger neurologic defect. Excision of neuromas and neurolysis seem to have no effect on the causalgic state and simply lead to more nerve stimulation with aggravation of symptoms

The Diabetic Neuropathy

While neuropathy has been recognized as a complication of diabetes for almost a century there is still much debate about its mechanism its incidence and its treatment Since arterial and arteriolar sclerosis is such a frequent concomitant of diabetes, pure diabetic neuropathy may only be seldom seen perhaps more often on the upper extremity or as a visceral form

The neurologic manifestations of diabetes mellitus may involve any part of the nervous system, namely the peripheral nerve trunks the spinal ganglia, the spinal cord, the autonomic nervous system and the brain The incidence of this lesion depends entirely on its definition in a recent monograph an incidence of 62 per cent is given of 261 cases studied [117] In studying our own peripheral vascular patients suffering from diabetes, we recognized it in almost every case, but this is obviously a selected group of patients

Sensory symptoms of numbness, tingling, paresthesia and burning are of course the symptoms of an ischemic neuritis many authors beginning with Woltman and Wilder [116] attribute diabetic neuropathy to atherosclerosis of the nutrient arteries of the nerve trunks But when one studies the upper extremities of some diabetic patients there is areflexia with such sensory symptoms and no demonstrable vascular lesion, neither in the periphery nor in the eyegrounds Such neuropathies must be due to the toxic metabolic effect of diabetes, perhaps to a thiamin deficiency In a middle aged diabetic physician who came to us with a sudden attack of pain and numbness in his upper and lower extremities and who was convinced that he had multiple emboli from an imaginary heart disease daily 50 mg. doses of thiamin chloride given intramuscularly for a week abolished all symptoms It is unusual however to see patients so early Usually when they are seen they complain either of a steady burning pain with paroxysmal aggravation not unlike a causalgic state or there is complete anesthesia of the sole of the foot together with anhidrosis a lack of Achilles tendon and occasionally of patellar reflexes Such a diabetic pseudotabes is often accompanied by

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diarrhea and bladder symptoms due to visceral autonomic neuropathy, so beautifully described by Rundles [118]

It is to be noted, however, that the vascular involvement and the neuropathy do certainly not parallel each other The hot, burning foot and the anesthetic foot with a trophic ulcer has fairly good circulation, there may even be pulses in the foot with some arteriolar sclerosis On the other hand, the gangrenous toes of the diabetic do not have prominent neurologic symptoms, except that the Achilles tendon reflexes are invariably lost.

Collens and his co-workers emphasized the impaired vibratory sense found in 90 per cent of the upper extremities and in 98 per cent of the lower extremities of diabetic patients [119] They felt that the diminished vibratory sense has no correlation with duration, severity and degree of control of the disease Our clinic has used a vibrometer, as devised by Collens and his group, both in the clinic and in the study of World War II veterans who suffered cold injuries We concluded, however, that the vibratory sense is diminished or abolished in many other situations not affecting the nervous system directly and is commonly absent after middle age [117] Nor could the vibrometer differentiate a metabolic from an ischemic neuropathy

The use of histamine flares as described in part II (p 58) reveals more than the impairment of cutaneous circulation Thus in many of the overwhelmingly neutrophic lesions the flares are positive unless the nerve lesion

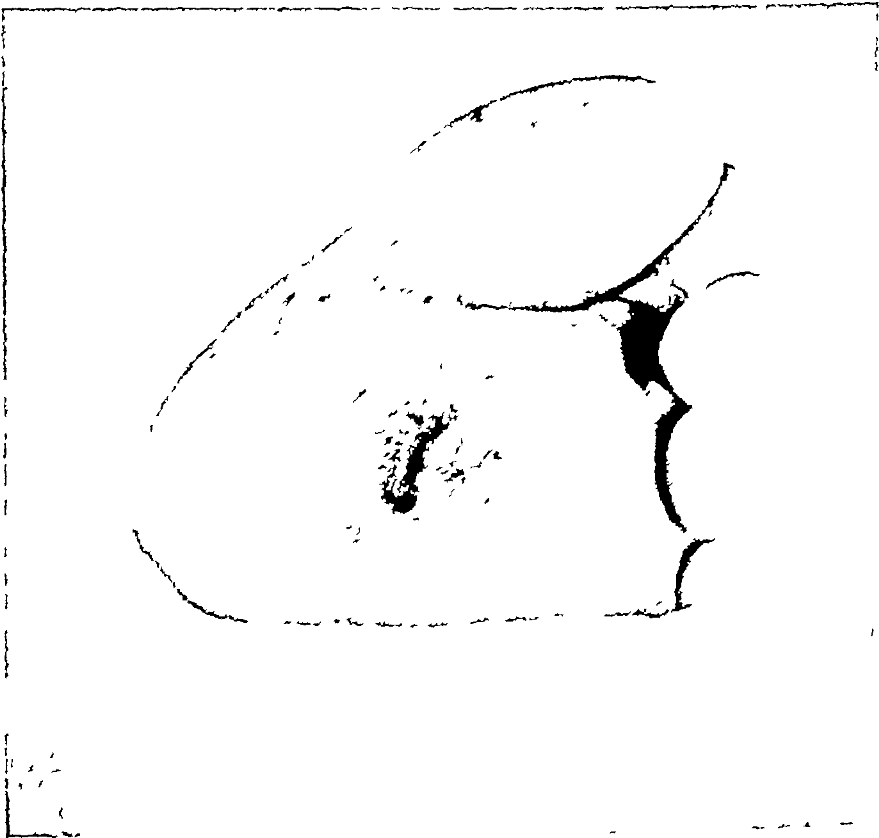


FIG 284 A trophic ulcer in a 53 year old diabetic patient, with absent patellar and Achilles tendon reflexes The ulcer was repeatedly infected and had been draining for over a year Removal of the second toe with its metatarsal head, excision of the infected callus with primary suture resulted in rapid healing

is so complete that anesthesia and a loss of axon reflexes are present Such a condition exists around a diabetic gangrenous heel or a completely painless malum perforans pedis of the sole of the foot On the other hand the painful hyperesthetic diabetic feet show a positive histamine flare but while the burning pain produced by the prick of intradermal histamine normally appears in three to four seconds it is delayed around involved trophoneurotic lesions to from 18 to 85 seconds This is just another expression of the well known fact that pain perception is delayed in some neurotrophic lesions and that the slowly conducting fibers are intact whereas the rapidly conducting myelinated pain fibers are damaged

For practical purposes we have recognized a type of neuropathy in which there is numbness tingling, burning pain and hyperesthesia such an extremity shows sympathetic activity often perspires and is greatly benefited by sympathectomy In a second group the dominant lesion is a trophic ulcer usually on the bottom of the foot, leading to a tarsometatarsal joint which may not show any bone destruction in the roentgen film but a probe gently inserted will find denuded bone The lesion is absolutely painless and the patient ordinarily walks on it (fig 284) Added to the recurrent ulcerations there are often large calluses, patches of hyperkeratosis and subcutaneous hemorrhages due to increased capillary fragility The crusts will fall off and



FIG 285 Trophic and hemorrhagic lesions in Frank LaM., who has had repeated admissions to the hospital for ascending lymphangitis. Skillful chiropody cleanliness, pHisoHex washings and large doses of hesperidin with vitamin C have kept him from losing any toes The feet are dry the neuropathy has sympathectomized him.



FIG 286 The diabetic gangrenous heel of Lorraine H, 53 years old, a well controlled diabetic woman Absolute bedrest, digestion of the slough with Varidase and a split thickness graft healed the lesion for five years

leave a good granulating surface Recurrent attacks of lymphangitis occur owing to poor foot hygiene, which can be best combatted by daily cleansing with Hexachlorophene G-11 (fig 285) Such feet are warm and dry and show no evidence of sympathetic activity The neuropathy here involves the sympathetics and the patient is sympathectomized [120] The trophic ulcer frequently is at the heel in diabetic patients, and this lesion is often painless and may be digested with Varidase (streptokinase-streptodornase), I have successfully grafted two of them (fig 286) The diabetic heel is frequently but not always painless and there is, of course, an element of arterial and arteriolar obstruction involved

In a third type of lesion, the neurotrophic bone and joint lesions, typical Charcot joints predominate The inadequate or absent sensation and the resulting motor instability and laxity must contribute to the amazing destruction, proliferation and subluxation of joints, together with a marked hyperemia These feet and ankles are warm, if not hot, and they do not require sympathectomies because they are sympathectomized (fig 287) Bailey and Root[121] described these "disorganizations" of the diabetic foot in detail and noted that the spinal fluid protein was elevated in 11 out of 14 patients Both they and Foster and Bassett[122] believe that the neurologic lesion affects the

posterior roots posterior root ganglia and the sensory components of the peripheral nerves with a relative sparing of the ventral roots and the motor fibers In such patients peripheral circulation is comparatively intact especially in younger individuals with severe diabetes one should adopt a radical attitude and if necessary do bilateral metatarsal amputations This was the case in a 34 year old severe diabetic patient who had multiple toe amputations and recurrent attacks of lymphangitis, and who actually developed an inflammatory lymphedema on the right side (fig 288) With the help of foam rubber packs in the toes of his shoes he can walk without difficulty

In addition to trophic lesions in the soft tissues and bone, occasionally a true onychogryphosis is seen in diabetic neuropathy (fig. 289) This patient with absent Achilles tendon reflexes hypesthesia of the foot and a subluxation of the second toe showed this hornified excrescence which was removed with the nail bed

Diabetic pseudotabes while rare has been seen in our clinic and the lancinating pain is out of proportion to the vascular and neurologic findings It is interesting from the vascular standpoint that in conjunction with the lancinating pain transitory increase in oscillations occur [123] and I have reported such a case in detail[124] (fig 290) This juvenile diabetic had a severe Charcot ankle on the right the curve was obtained during an attack of paroxysm This is all the more striking since radicular pain, as seen in osteoarthritis of the spine is usually accompanied by vasoconstriction



FIG 287 Spontaneous fractures, trophic joints, and amputation of big toe in a diabetic patient with trophoneurosis. Midmetatarsal amputation healed well.



FIG 288 Bilateral midmetatarsal amputation in severe Charcot joints of a 34 year old diabetic radio announcer There is brawny induration and edema, especially on the right because of multiple attacks of lymphangitis Note the incision on left for osteomyelitis, which healed well



FIG 289 A hornified nail of the right big toe, some of it is also visible on the fifth toe There was a diabetic ulcer over the right ankle, loss of Achilles tendon reflex and hypesthesia of the foot

To explain these bouts of painful vasodilation one cannot help but think of ischemic or apoplectic lesions in the posterior root ganglion which is nourished by a small, thin twig from the intercostal artery [125] It was pointed out from our clinic that certain diabetic patients with atheromatous plaques of the aorta at the origin of intercostal arteries have ischemic or apoplectic spinal ganglia which can be histologically demonstrated [126] The firing of impulses from these ischemic posterior root ganglia and the resulting paroxysmal pain and vasodilation constitute the same problem as a tabetic crisis No type of sympathectomy relieves such pain but high and unilateral antero-lateral cordotomy is promising in experienced hands [8]

Treatment

I have already referred to the fact that the different modalities of diabetic neuropathy require selective methods of treatment Certainly if the vascular lesion is prominent and if major arterial occlusions are present, they should be treated as any other form of segmental or diffuse arterial sclerosis Arteriolar sclerosis is of course not amenable to surgical therapy and here nicotinic acid and its alcoholic derivative Roniacol are helpful In the early metabolic neuropathy large doses of thiamin chloride 50 to 100 mg daily or vitamin B_{12} in 1 mg. (1000 μg) doses may be tried but if ischemic neuropathy with involvement of the vasa nervorum is present,[126] such medication is bound to fail For the capillary fragility hesperidin with vitamin C is useful

Most diabetic neuropathies as has been pointed out, have adequately sympathectomized themselves and sympathectomy is unnecessary The

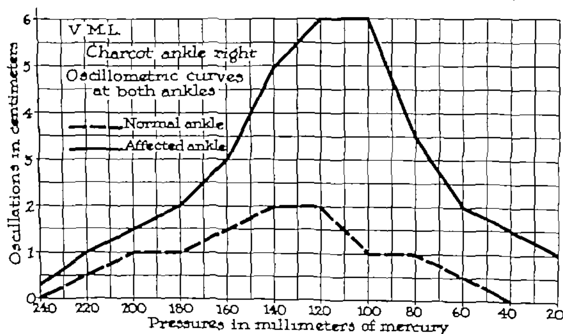


FIG 290 Oscillometric curves at the left and right ankles of a 29 year old juvenile diabetic with severe diabetic neuropathy including a Charcot ankle on the right. During an attack of paroxysmal pain, this tracing was obtained, not unlike one obtained in the vasodilator phase of causalgia.

trophic lesions, including those of the toes or heel, can usually be safely excised and covered, this is necessary because of the recurrent attacks of spreading cellulitis and lymphangitis It is mostly in these cases that the metatarsal amputation for diabetic gangrene of the toes, so warmly advocated by McKittrick and his co-workers,[127] is successful in our hands

The neurotrophic foot, whether draining or closed, should be daily washed with pHisoderm and adequately protected against undue pressure, infection or exposure to cold Sympathectomy in the hot, burning, paresthetic leg helps the causalgic state by selective degeneration of some sensory fibers and with intact sympathetics For the lancinating pseudotabetic crises, sympathectomy is ill advised and neurosurgical consultation should be sought In my experience, neurosurgeons are hesitant to do anterolateral cordotomies in patients with arteriosclerotic cords

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CHAPTER 17

THE CAROTID SINUS SYNDROME

FUNDAMENTAL CONSIDERATIONS

AT THE BIFURCATION OF THE CAROTID ARTERY THERE ARE A NUMBER OF SENSORY end organs which continue into visible nerve trunks and plexuses and send afferent fibers to the ganglion nodosum of the vagus and the glossopharyngeal nerve (fig 291) The carotid sinus also receives efferent fibers from the

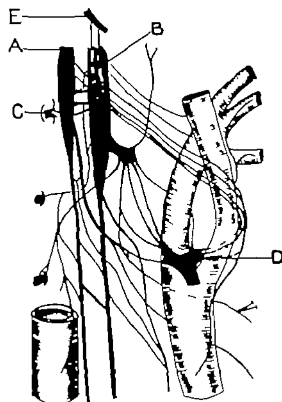


FIG 291 (A) Ganglion nodosum of the vagus nerve (B) The superior cervical sympathetic ganglion (C) The second cervical root and origin of the superior laryngeal nerve (D) The carotid sinus with connections to the glossopharyngeal nerve. (E) The fiber from the superior laryngeal nerve (Modified from Delmas, J and Laux, G Anatomie Médico-Chirurgicale du Système Nerveux Végétative Masson et Cie, 1933)

superior sympathetic ganglion. Hering[1] pointed to the cardiovascular and respiratory reflexes originating from the carotid sinus and this work was summarized and greatly expanded in a monograph by Heymans Bouckaert and Regniers [2] In the same year (1933) Weiss and Baker[3] described a hitherto ignored syndrome of recurrent attacks of syncope due to a hyperactive carotid sinus reflex Thus in the early 1930 s a great interest began in this area which lately has lost much momentum perhaps due to the fact that many patients originally thought to have had a hypersensitive carotid sinus

reflex have other cardiovascular defects and this syndrome is only a secondary phenomenon

Ask-Upmark[4] has provided us with extensive investigations on the comparative anatomy of the carotid sinus nerves He studied 61 animals representing 27 different species of mammals, birds, reptiles and amphibia Wherever an internal carotid artery was present, the carotid sinus was there originating from the bifurcation In species where the internal carotid artery was absent, the sinus nerves appeared at the base of the occipital artery Ask-Upmark felt that the carotid sinus reflex was a protective mechanism, insuring even blood flow to the brain by buffering fluctuations of pressure.

The sensory nerve endings have the typical structure of stretch receptors, such as those found in the aortic arch and in tendons The afferent fibers course mainly through the glossopharyngeal nerve, and when this nerve is cut for glossopharyngeal neuralgia it is liable to produce a sudden fluctuation of blood pressure, unless these are suppressed by the depth of anesthesia

There are a number of factors which influence carotid sinus reflexes During the conduction of anesthesia, the reflexes maintain a compensatory sympathetic adrenal discharge to stabilize blood pressure, only in the deepest, fourth stage of anesthesia is the reflex abolished The fall in blood temperature, as produced in hypothermia, stimulates the carotid sinus to prevent a fall in blood pressure, however, the use of the "lytic cocktail" (see hypothermia, p 540), notably Thorazine and Phenergan, dampens these reflexes so that a controlled hypotension may ensue

A rise of pressure in the carotid sinus produces reflexly (1) a slowing of the heart, (2) a widespread vasodilatation and consequently a fall in blood pressure, (3) a diminished secretion of epinephrine, (4) a depression or arrest of respiration, (5) a diminished tone in skeletal muscle, and (6) visceral changes, such as an increased stomach tone and decreased bladder tone Conversely, when the pressure in the sinus is lowered the reverse effects are produced, *i e*, cardiac acceleration, increased cardiac excitability, extrasystoles, vasoconstriction, a rise in blood pressure, an increase in the depth and rate of respiration, an increased secretion of epinephrine and changes in gastrointestinal tone [5]

In addition to the carotid sinus nerve, the carotid body lies at the carotid bifurcation and functions as a chemoreceptor (fig 292) It is affected by alterations in CO_2 and O_2 tension, by H^+ ion concentration and by certain drugs, such as nicotine, acetylcholine and cyanide The afferent impulses arising in these bodies reflexly modify respiration If excess CO_2 is present, respiration is markedly stimulated There are also reflex effects on the circulation, such as acceleration of the heart and blood pressure Particularly, hemorrhage and anoxia stimulate the chemoreceptors

Besides the carotid body, the aortic body is another active chemoreceptor. The carotid body receives an abundant blood supply, however, if this is cut off or blocked, the baroreceptors in the carotid sinus still function but the chemoreceptors cannot This can be demonstrated in a species

of animals in which the carotid body receives its blood supply from the occipital artery

In such animals whose carotid sinus and aortic reflexes have been eliminated anoxia causes not a rise but a fall in blood pressure This is significant, since in deep anesthesia the usual signs and symptoms of anoxia or hypercapnia may be missing.[6]

When norepinephrine is applied to the wall of the sinus, a marked reflex fall in blood pressure occurs This has been explained as being due to a change in tone and tension of the barosensitive arterial segment[7] Electrocardiograms and electroencephalograms can readily be obtained during the production of the carotid sinus syndrome

CLINICAL MANIFESTATIONS

Three types of carotid sinus reflex have been described (1) the *vagal type* producing bradycardia or asystole which can be interrupted with atropine (2) the *depressor type* resulting in a marked fall in pressure without much bradycardia and counteracted with pressor substances such as epinephrine norepinephrine or Neo-Synephrine and (3) the *cerebral type* in which fainting or convulsions occur with or without marked changes in heart rate or blood pressure This primary cerebral effect can only be treated by procaine infiltration of the carotid sinus, or by denervation[3]

In addition to the full blown syndrome which is not too frequent, there is a large number of patients who have a hyper reactive carotid sinus secondary to some factor facilitating this reflex Digitalis morphine and anoxia increase vagal irritability and may produce striking changes in cardiac conduction such as heart block temporary asystole nodal rhythm, ventricular extrasystole changes in the T waves and complete inversion of the electrical

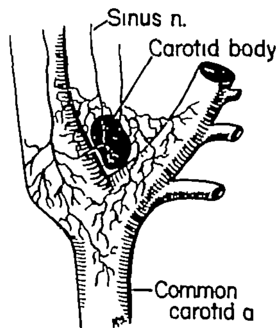


FIG 292. The carotid body is essentially a chemoreceptor Removal of its tumor is usually possible without injury to the external or the internal carotid artery

axis Small lymph glands adhering to the carotid bifurcation produced an increased irritability in one of our own cases The origin of the internal carotid may show a bulbous dilatation or may be sclerotic, stenotic or occluded, all of which have been seen to contribute to a sensitive sinus A sensitive carotid sinus mechanism was observed in a patient with an aortic arch syndrome (p. 465) Cerebral arteriosclerosis seems to increase the irritability of the mechanism, probably through anoxic stimulation of the central receptors

TEST FOR HYPERACTIVITY OF THE SINUS MECHANISM

A pressure point on the neck causing dizziness, faintness or convulsions has been recognized ever since Cermak (1866) palpated a small nodule on the right side of the neck This was thought to be a vagus effect for a long time, and it was mostly through the observations of Weiss and Baker[3] that the carotid sinus was made responsible for this phenomenon The reflex may be elicited by direct pressure on the carotid bulb in the sitting position, but *never pressing on both sides at once.* Or, one can obliterate the common carotid artery below the bifurcation and elicit the reflex on release of the compression, the incoming pulse wave suddenly distending the carotid sinus

In normal subjects, pressure on the carotid sinus causes a drop of less than 10 mm of mercury and the bradycardia is slight An exaggerated reflex is present if marked bradycardia, definite hypotension or faintness and convulsions develop

The sinus is especially irritable in older arteriosclerotic individuals Pressure on the neck, occurring on hanging, choking and during boxing, results in unconsciousness During general anesthesia, the anesthetist may press on one or both sinuses in holding the jaw forward In removing cervical lymph glands, operating on the thyroid gland, removing a carotid body tumor and during cerebral angiography, opportunities arise for the stimulation of this mechanism Infiltration of procaine into the area of carotid bifurcation is an effective safeguard against such reflex phenomena Ether anesthesia may dampen such reflexes, but only if it is deep, whereas Pentothal sodium, nitrous oxide or cyclopropane do not dampen these reflexes [8]

The hyperirritability may be limited to one side and very little disturbance is elicited from the opposite one Putting on a stiff collar, turning the head suddenly and hyperextending the neck during some surgical or dental procedure have been reported as bringing on alarming syncope

Since an irritable sinus may manifest itself in convulsive seizures, in heart block and in other vagal effects on the heart, the mechanism should be tested routinely in individuals showing such symptoms

TREATMENT

Mild irritability of the sinus, manifesting itself in vagal or depressor effects, can be treated—at least for a trial—with atropine or other parasym-

pathetic depressants,[9] and with ephedrine or Benzedrine if the hypotension is prominent However in our experience carotid sinus denervation on one or both sides is a specific cure for this syndrome provided the syndrome is really present and the indication for the operation is correct

No patient should be considered for denervation of the carotid sinus unless his symptoms can be reproduced by stimulation of the carotid sinus and unless the mechanical stimulation is ineffective after the sinus has been infiltrated with procaine The technique of this injection has been carefully described by Pick and Wertheim [10]

The literature on the effects of denervation of the carotid sinus has been admirably dealt with in the monograph on the autonomic nervous system by White Smithwick and Simeone [11] Our own experience which includes two cases of bilateral and three cases of unilateral denervation has been gratifying. In two patients, R B Capps and I[12] found that no permanent hypertension or tachycardia results in man from bilateral denervation of the carotid sinus At the time of study (8½ and 17 months after operation) both patients failed to show a rise in blood pressure or in pulse rate on exercise comparable to that of normal controls On the other hand both showed a marked postural hypotension, one of them fainting after two minutes In both patients there was considerable hypertension (152/100 mm Hg and 146/100 mm Hg) at their discharge from the hospital, but the duration of this temporary hypertension is limited Undoubtedly the aortic depressor mechanism is able to assume this function of the carotid sinus but the presence of postural hypotension would indicate a lower sensitivity of the aortic depressor mechanism in elevating blood pressure [13]

In another case of unilateral carotid sinus denervation done under local anesthesia, blood pressures were recorded during several phases of the operation with a Tycos recording sphygmomanometer (fig 293) Manipulation of the carotid artery raised the blood pressure from an initial level of 138 80 mm Hg to 180/110 mm Hg. This may have been due to a sensory stimulation, although the patient did not register any pain Anesthesia of the sinus with ½ per cent procaine resulted in a spectacular rise in pressure to 215 110 mm Hg. As dissection was continued, marked cardiac irregularity was noted probably due to stimulation of the vagal fibers On complete removal of the sinus nerves, the huge pulse waves and the hypertension persisted, but by closing of the skin the pressure had dropped to 180/90 mm Hg Two months later the blood pressure was 112 70 in the recumbent and 112/80 mm Hg in the standing position showing the normal slight pressor response of the erect posture.

It can be said then as we pointed out in 1938 [12] that bilateral carotid sinus denervation, although it does not result in permanent hypertension does deprive man of an important compensatory mechanism of erect posture in that a postural hypotension may develop This does not occur on unilateral denervation However since neither our two patients nor those at the Massachusetts General Hospital who have had bilateral denervation had any clinical symptoms of postural hypotension, the bilateral operation is

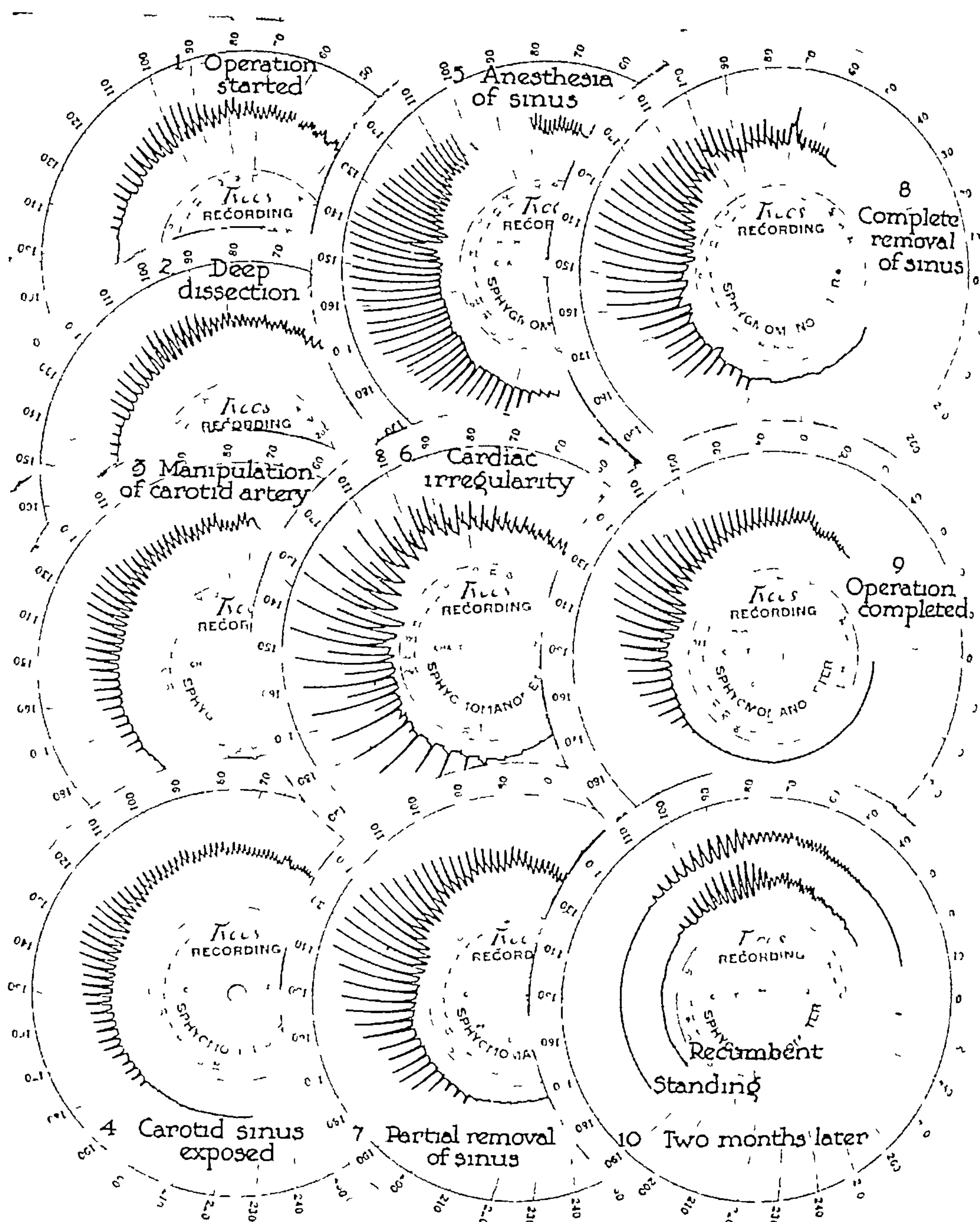


FIG 293 Blood pressure records during denervation of the carotid sinus under local anesthesia Note the marked rise in blood pressure and increased height of pulse waves in (5) when the bifurcation was infiltrated with procaine Also note the cardiac irregularity appearing on dissection in (6), probably due to traction on sinus nerves In (10), two months after the operation, there is no hypertension and no postural hypotension

justified if evidence of bilateral sinus irritability is present The sinus denervation for certain cases of epilepsy as attempted by Wilder Penfield and also by us has not stood the test of time and has been generally abandoned

In one patient, Margaret T , who complained of an atypical facial neuralgia, pain in the jaw and a unilateral throbbing headache all on the right side, a right cervical sympathectomy was done in 1937 which gave her short relief In 1940 it was found that she had a bilateral hyperirritable carotid sinus, more pronounced on the right side, which produced faintness, hypo-

tension and black spots in front of her eyes on direct pressure The right carotid bifurcation was exposed and after the operative scar was dissected away a fusiform aneurysm of the internal carotid artery was visualized at the level of the sinus Because of the heavy scarring and some calcification it was thought better to reduce the aneurymal tension by slow ligation of the common carotid artery The braided silk ligature was left long so that it could be quickly released if necessary but no cerebral symptoms developed The patient was completely relieved of her symptoms and when re-examined in 1956 16 years later the result still persisted Since her opposite sinus also showed some sensitivity a congenital weakness of the bulb may have been present Common carotid ligation is not advocated ordinarily for the carotid sinus syndrome but in this case it effectively abolished the patient s symptoms

Procaine infiltration of one or both carotid sinuses for the treatment of acute neurogenic hypotension has created quite a stir in France Arnulf[14] has given a complete review of this subject and feels that in acute shock which does not respond to the usual measures it might produce a sudden massive stimulation of the adrenals and an occasional surprising rise in blood pressure I have had no personal experience with this method

In removing chemodectomas tumors arising from the carotid body it is usually possible to remove the entire mass by stripping the adventitia with it and not to injure either the external or the internal carotid artery [15] A lateral suture of the internal carotid or a patch of vein or artery placed into an accidental rent, should always be tried before ligation is done While carotid body tumors occasionally metastasize they generally will not recur on removal in toto [16] Naturally the carotid sinus is denervated in this procedure and is often hypersensitive before operation

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CHAPTER 18

THE AORTIC ARCH SYNDROME

DEFINITION

THE AORTIC ARCH DELIVERS BLOOD THROUGH ITS BRANCHES TO THE HEAD TO the neck and to both upper extremities When the branches of the arch be come narrow or obliterated a peculiar disease entity develops called pulseless disease Takayasu s disease reverse coarctation of the aorta or Martorell s syndrome The nomenclature of Frøvig,[1] aortic arch syndrome seems most appropriate * This is defined as an absence or diminution of pulses in the arms and neck, and is of multiple etiology

ETIOLOGY AND PATHOLOGY

Congenital Anomalies

It is likely that an aortic arch may be aplastic or nonpatent, but such a condition would not be compatible with life More important are the unusual arrangement of vessels supplying the head neck and arms De Garis and his co-workers[3] have described five basic patterns Two vessels may arise from the arch by a common trunk or merely by a common ostium In 1930 Kampmeier and Neumann[4] suggested that the anomalous origin of vessels may render them more susceptible to occlusion and they presented such postmortem specimens even though the basic lesion was a luetic aneurysm of the aorta

No discussion of aortic rings, the right aortic arch or other congenital anomalies of the aorta need be pursued here since they are not accompanied by changes in the pulse in the two arms Coarctation and patent ductus arteriosus, however may give rise to such findings Weak or absent pulse in the left arm may be present in coarctation when the left subclavian artery is involved The coarctation may even occur proximal to the origin of the left subclavian artery In addition, the right subclavian artery may be involved by stricture

In cases of patent ductus arteriosus, there may be unequal radial pulses with a higher pressure in the right than in the left arm Oda[5] found this to be true in all of the seven cases of patent ductus arteriosus which he studied

* However priority for recognizing and describing this syndrome should go to Martorell and Fabre Tersol[2] who described the obliteration of supra-aortic branches in a Spanish communication unknown to Frøvig.

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CHAPTER 18

THE AORTIC ARCH SYNDROME

DEFINITION

THE AORTIC ARCH DELIVERS BLOOD THROUGH ITS BRANCHES TO THE HEAD TO the neck and to both upper extremities When the branches of the arch become narrow or obliterated a peculiar disease entity develops called pulseless disease, Takayasu s disease reverse coarctation of the aorta or Martorell s syndrome The nomenclature of Frøvig,[1] aortic arch syndrome seems most appropriate * This is defined as an absence or diminution of pulses in the arms and neck and is of multiple etiology

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It is wise to examine every patient for difference in pulses and blood pressures in his two arms The long-standing Oriental interest in the pulse has led to many publications on this syndrome in China and Japan. Caccamise and Whitman[6] found 58 cases of the aortic arch syndrome reported from Japan How many of these had a congenital basis—even though an arteritis were superimposed on it—is unknown.

Trauma to the Arch

Because of the great increase in steering-wheel injuries and the medicolegal implications, one has to consider both an aneurysm of the arch and occlusion of the vessels of the arch following severe trauma to the sternum. The left subclavian ostium seems especially vulnerable, and it is here where the aorta is more apt to rupture.[7] There are a number of case reports in the literature in which the connection between a single, massive trauma to the chest with a subsequent development of aneurysm, rupture or partial obliteration is clearcut These have been summarized in the excellent survey of Ross and McKusick [8] In such cases, the blood pressure and pulse are often markedly different in the two upper extremities

Just as in peripheral arteries, trauma may bring on an acute, obliterating thrombosis in a previously diseased artery In a case observed at Johns Hopkins Hospital,[8] a known syphilitic patient held his neck in prolonged hyperextension during the course of dental extractions Pain in his right eye developed within the next few hours and he was blind next morning in the right eye, with hemiplegia on the left. His common carotid artery was thrombosed at its origin from the aorta

We saw a 59 year old hypertensive school teacher, who presented chest films of a normal thoracic aorta prior to a steering-wheel injury and who then developed a steadily increasing dilatation and later an aneurysm of the arch. There was marked difference in pulse and blood pressure in her two arms, which gave rise to the erroneous diagnosis of peripheral vascular disease It is also possible for aneurysms of any type to throw emboli into peripheral vessels and this we have observed several times

Trauma may also result in intimal tears, thromboses or stenoses in the aorta as in other vessels, even atheromata may develop at the site of vascular injury The connection of trauma with this type of disease, however, is always difficult to prove

Syphilitic Aortitis

Of 547 patients suffering from cardiovascular syphilis, 309 had aortic insufficiency, 95 had aneurysms, 2 had coronary ostial stenosis and 141 exhibited syphilitic aortitis [9] Early vascular syphilis cannot always be accurately diagnosed, but the roentgen ray evidence of a widened aorta, the accentuated aortic second sound, the submanubrial dullness and the systolic murmur at the aortic area are important diagnostic leads

15 per cent of such untreated patients develop aortic insufficiency or aneurysm after an average of 3 5 years Often the *inequality of radial pulses* first suggests to the clinician the presence of an aneurysm, and as far back as 1628 Harvey in *Exercitatio anatomica de motu cordis et sanguinis* described a case in which "the pulse in the corresponding arm was small in consequence of the greater portion of the blood being diverted into the tumor and so inter cepted. Nevertheless, this is not a useful or usual sign since Hare and Holder[10] found abnormalities of the pulse in only 32 cases out of 953

Occlusion of one or several vessels of the aortic arch in cases of a luetic aneurysm may occur as a result of the stenosis or obliteration of their origin due to the inflammatory process or due to actual mural thrombi Ross and McKusick[8] have published several postmortem studies where the ostia of these vessels were occluded by gray fibrous tissue or thrombi A diffuse stenosis of the arch is hardly if ever present in luetic aortitis

Nonsyphilitic Aortitis

In a small group of patients obstruction at the origin of the large ar teries of the arch of the aorta occurs in the presence of well defined vascular disease such as thromboangiitis obliterans panarteritis nodosa or temporal arteritis In Gilmour s[11] cases of giant cell arteritis there was, in addition to the involvement of the temporal arteries an occlusion of the common carotid arteries and of the left subclavian artery and in one case an obstruc tion of the innominate artery In a case seen in consultation a left sided temporal arteritis was accompanied by a tender thickened and poorly pulsat ing common carotid artery on the same side A few months later the axillary pulse was lost on the same side presumably due to obstruction at the ostium of the subclavian artery The true nature of this chronic spreading granu lomatous arteritis is unclear

A summary of cases of the young female arteritis type of aortic arch syndrome is given by Ross and McKusick [8] These are all young women, showing no evidence of syphilis rheumatic infection or atheromatosis To- gether with loss of radial pulses, they develop attacks of dizziness, visual symptoms, transitory paralysis and hemiatrophy of the face with twitching. Grayish thrombi or gelatinous masses fill the origin of the innominate the left subclavian and the common carotid arteries Histologically there are in flammatory reactions around the vasa vasorum and in the adventitia, more like a periarteritis or an endarteritis obliterans Most women are between the age of 20 and 35 years

We have recently seen a young woman with a cold pulseless right hand exhibiting a continuous murmur over the right sternoclavicular joint This murmur was transmitted toward the heart and was audible in the left cubital fossa. She refused hospitalization for angiocardiographic study Aortic stenosis or hypoplasia, however may give rise to this murmur since five patients of the young female series exhibited it One wonders of course

about the possibility of multiple vascular anomalies here In the case observed by us, a deformed hypoplastic hand was also present

Atheromatosis

There are a number of cases in the literature in which the ostia of the carotids or of the innominate and subclavian arteries are plugged by atheromatous plaques Syphilitic aortitis renders the aortic arch more vulnerable to atheromatosis. Certainly, pure atheromatosis is much more frequent in the abdominal aorta, and when it appears in the aortic arch some predisposing factor, such as a congenital vascular anomaly or syphilitic aortitis, may constitute the localizing element Atheroma at the carotid bifurcation has already been discussed on page 207

One sees a goodly number of patients with atheromatosis of the bifurcation of the abdominal aorta, who show a marked difference in pulse and blood pressure in the arms While I have no statistical data to submit, it is our standing rule to watch for this difference in pulses at the wrists, especially if the lipid metabolism is abnormal, as in nephrosis or xanthomatosis Raynaud's syndrome in arteriosclerotic patients is occasionally caused by subclavian or innominate arterial stenosis at their origin from the aorta.

Dissecting Aneurysm of the Arch

We are not concerned here with the acute fulminating dissections which cause death within a few days, but we need to consider healed dissections which obliterate the origin of the vessels of the arch In Shennan's review of 287 cases, the inequalities of the radial pulses are mentioned [12] Vessels may be dissected for a considerable distance from the aortic arch, the occlusion may be so far from the origin of the vessel that the process may be mistaken for an embolus (fig 294) Such was the case in a middle-aged hypertensive woman seen in consultation who, after an excruciating chest pain which was thought to be a coronary occlusion, lost her pulses first to both upper extremities and then to one lower extremity She was in too critical a state to consider embolectomy On autopsy, the dissection occluded the innominate, the subclavian and the left common iliac arteries This mechanism should be thought of in cases of acute arterial occlusion

CLINICAL SYMPTOMS

The clinical picture is dependent on the decrease of blood flow through the arterial channels originating from the arch of the aorta The syndrome of common carotid thrombosis is not as pronounced as that of the occlusion of the internal carotid artery, of which 107 cases have been collected by Johnson and Walker [13] This syndrome has recently been discussed from our service in connection with the emergency treatment of apoplexy,[14] and is not identi-

cal with the occlusion of the common carotid at the aortic stoma unless a thrombosis occludes the carotid bifurcation

Vertigo on rising from the horizontal position short periods of loss of consciousness, hemiparesis or hemiplegia convulsions severe unilateral headaches, transient periods of blindness dimness of vision associated with exercise (referred to by Frøvig[1] as visual claudication) and a surprising incidence of cataracts have been reported In a case observed in the Vascular Clinic of the University of Illinois College of Medicine hemiatrophy of the face with muscular twitching, claudication of the jaw muscles on mastication and dental pain for which extractions were done were in evidence

With the ophthalmoscope optic atrophy a decrease in retinal arterial pressure and wreathlike peripapillary anastomoses may be seen [15] It is interesting to note however that because of the free collateral circulation between the external carotid and ophthalmic arteries of the same side only about 10 per cent of Johnson and Walker s collective cases showed blindness

Changes in hearing are not so prominent as the symptoms referable to the eye however deafness or tinnitus have been occasionally reported

Sites & Extent of Dissecting Aneurysms of the Aorta-287 cases (Shennan)

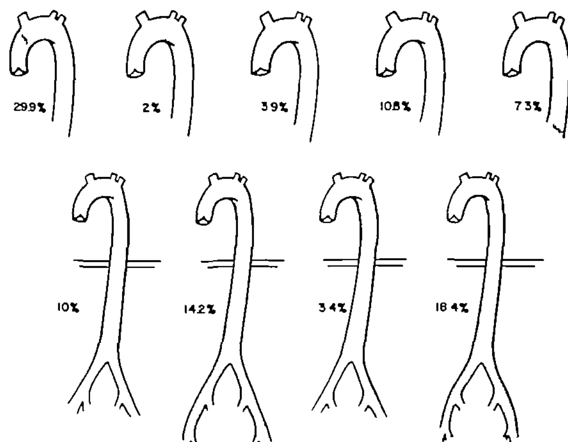


FIG. 294 Extent of dissection of 287 collected cases of dissecting aneurysm of the aorta (Shennan, T Dissecting Aneurysms. Medical Research Council, Special Report Series No 193 His Majesty's Stat. Off., London, 1934)

Evidence of vascular insufficiency in the arms and hands is infrequent. The arm with the feeble or absent pulse may be cooler, and occasional numbness, pallor or cramping on exercise is noted. Generally speaking, however, a satisfactory collateral circulation develops during the period of a slow occlusion of the innominate or left subclavian artery, and the patients may not be aware of any ischemia in the upper extremities

A sensitive carotid sinus mechanism has been reported in seven patients,[8] manifestating itself by a fall in blood pressure, by bradycardia, by a loss of consciousness or by hyperpnea on carotid sinus pressure or on sudden rising Since partially occluded arteries produce systolic murmurs proximal to the obstruction, these can be looked for and sometimes heard at the base of the neck, left or right of the midline. Occasionally a machinery-like murmur may be heard Confusion with a patent ductus arteriosus may occur

DIAGNOSIS

An adequate history may reveal chest trauma, a history of syphilis or symptoms of carotid or subclavian arterial insufficiency The presence and intensity of the carotid pulses are ascertained. Pulses and blood pressures are taken in both arms and auscultation at the base of the neck is done for the presence of bruits Trophic changes in the eyes or nose are looked for and the patient is tested for a sensitive carotid sinus. Tests for syphilis, for blood lipids and for an abnormal clotting tendency are performed

Chest roentgenogram and fluoroscopy may reveal abnormal changes in the aorta or in its major branches Aortograms have been performed on our service either in a retrograde fashion with a catheter in the femoral artery or, preferably, directly through the aortic arch (p 65) It is difficult to visualize the occlusions, and exploration may be necessary to decide on operability, especially regarding the patency of the subclavian or carotid arteries distal to the obstruction

DIFFERENTIAL DIAGNOSIS

There are a number of other causes of loss or diminution of pulsation in the neck or upper extremity Only cases in which the occlusion is at or near the origin of the artery from the aorta truly belong to the aortic arch syndrome Trauma to the vessels of the neck or axilla, thromboangiitis obliterans and embolic occlusion of carotids or subclavian vessels can usually be ruled out. Peripheral arteriosclerosis affecting the carotid arteries or the branches of the brachial artery is not a part of the syndrome. The syndrome of the thoracic outlet, including a cervical rib, scalenus syndrome and hyperadduction syndrome have a distinct symptomatology

TREATMENT

If syphilitic aortitis is diagnosed, antisyphilitic therapy is in order, first with bismuth and iodides then with massive doses of penicillin Two courses

of penicillin treatment given six weeks apart can be administered giving 600 000 units daily intramuscularly for 10 days at a time In at least three cases the pulses have been restored by this treatment [8] but even if this does not happen it is probably wise to precede any vascular suture with antisyphilitic therapy so as to prevent a local granulomatous inflammation at the operative site

When lues can be excluded thromboendarterectomy and vascular grafts may be considered Gordon Murray (cited in 8) has reopened a closed common carotid artery by reaming it out from the neck toward the arch Henry Bahnson (cited in 8) grafted an infant s lyophilized aorta between the arch and the patent distal segment of the common carotid artery and relieved the patient s dizziness tinnitus and convulsions In our institution Davis Grove and Julian[16] reported on a 51 year old patient whose syncope and convulsive twitchings were relieved by endarterectomy of the innominate artery However the subclavian pulse was not restored This patient was followed in the vascular clinic with a Leriche syndrome and progressive diffuse atheromatosis

DeBakey and his associates[17] reported two cases one was relieved by endarterectomy at the level of the carotid sinus followed by the implantation of a bifurcated Nylon tube from the aorta to the subclavian and common carotid arteries In the second case a left subclavian artery was reamed out Normal pulsations were obtained in both cases and both patients were relieved of their symptoms

It was pointed out by Da Costa and Fagundes[18] that the fully developed syndrome is easily diagnosed, but the early phase of the process may be a unilateral or bilateral subclavian stenosis and this incomplete Martorell s syndrome may well be amenable to endarterectomy It is well conceivable that by relieving this localized stenosis the development of the full syndrome particularly the involvement of the carotid arteries may be prevented

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CHAPTER 19

ESSENTIAL HYPERTENSION

THE INCLUSION OF THE SUBJECT OF HYPERTENSION IN A MONOGRAPH ON vascular surgery is justifiable on two counts First, essential hypertension no matter how defined becomes an arteriolar disease after a phase of increased peripheral resistance Second our vascular clinic has had a long time in tensive interest in this subject, and we can look back on this problem now with a sufficient number of surgically treated patients observed for 10 to 20 years [1]

Of course it will not be possible here to review or discuss all of the facets which this subject presents Exhaustive and up to date monographs are available,[2] and any surgeon becoming interested in the surgical treatment of hypertension must be familiar with the experimental methods of producing hypertension and with the results of treatment by diets drugs and surgical procedures, so that he can decide on the optimal course to be taken in each individual patient In this chapter I will especially dwell on the selection of cases for operation the surgical techniques will be described in part IV of this volume

DEFINITION

Essential hypertension is mainly a diagnosis of exclusion made by the clinician with the help of a sphygmomanometer with or without recognizable arteriolar lesions [2f] It is not the arteriosclerotic hypertension with a low diastolic and a high pulse pressure so often mistaken for essential hyper tension and so overtreated by many clinicians It is not coarctation of the aorta at typical or atypical levels which readily reveals itself by a gentle touch at the groin for a delayed or diminished femoral pulse It is not an obvious renal or endocrine hypertension All of these entities will be dis cussed under differential diagnosis (p 502) It is customary today to regard any patient in whom the above primary causes can be excluded and who on repeated examinations exhibits a blood pressure of 150/100 mm. of Hg, as suffering from essential hypertension Obviously with increasing recognition of etiologic factors, more and more clinical entities will be separated from "essential hypertension

Essential hypertensive patients according to Pickering,[2c] represent no more than that section of the population having arterial pressures above a

certain level, selected on arbitrary grounds, and having no disease to account for these arterial pressures "

MECHANISMS OF HYPERTENSION

The experimental production of chronic hypertension in animals has greatly helped in understanding some of the factors which are responsible for the elevation of blood pressure *Neurogenic hypertension* was produced by Heymans[3] by denervation of the carotid sinus and section of the aortic depressor nerves This type of hypertension can be abolished by total paravertebral sympathectomy In our own experience reported with Richard B Capps,[4] bilateral carotid sinus denervation with intact aortic depressor nerves results in only temporary elevation of blood pressure, but in a significant postural hypotension (p 461) Rigidity due to atherosclerosis and the aortic arch syndrome, resulting in obstruction of the common carotid artery, increases the sensitivity of the carotid sinus (p 470) Placing a lucite cast around the carotid bifurcations, Wakerlin and his associates have produced experimental hypertension in dogs[5] Also, an acute rise in intracranial pressure, induced by the intracisternal injection of kaolin, may be abolished by sympathectomy[6] All these experiments simply mean that centrally or reflexly induced vasoconstrictor outflow or a cerebral mass or substance can produce hypertension and may be abolished by extensive paravertebral sympathectomy

Attempts to correlate this experimental, buffer-nerve hypertension with human, neurogenic hypertension have, naturally, been made Especially Heymans[7] has investigated the state of contraction, tone, tension and distensibility of the arterial wall of the sino-aortic areas A whole number of vasoconstrictors applied locally to the carotid sinus provoke a marked reflex fall in blood pressure, whereas smooth muscle relaxants, such as papaverine and benzylimidazoline, when applied locally will induce a reflex rise in blood pressure From his experimental work, Heymans concluded that the decrease in tone and decreased resistance to stretch could be the primary mechanism of hypertension

The difficulty of the clinician in accepting this mechanism to be operating in essential hypertensive patients, lies in the often made observation that the carotid sinus reflex is present and seems normal in essential hypertension Our group has tested this reflex for many years in all preoperative patients and could not find any abnormal, diminished response It may be that this is a crude functional test and that the findings of Kezdi,[8] in which the procaine block of the carotid sinus elicited less of a rise in blood pressure in hypertensive than in normotensive patients, may have to be repeated Shortly after our observations with Capps,[4] that bilateral carotid sinus denervation produced postural hypotension in normotensive patients, two hypertensive patients who had rising blood pressures after a transdiaphragmatic splanchnicectomy were subjected to cervical sympathectomy with carotid sinus denervation No additional improvement to that already obtained was noted.

Another interesting method of producing neurogenic hypertension is to stimulate the central end of the vagus sectioned in the neck which effect is abolished by cutting the cervical spinal cord or by total sympathectomy Adrenalectomy does not abolish this hypertension This type of hypertension in which afferent visceral stimuli activate the sympathetic nervous system may well be based on the production of epinephrine and norepinephrine in the arterial wall the interested reader is referred to the often quoted Ciba Symposium on Hypertension especially the work of the Madrid investigators [7,8]

Another important type of experimental hypertension is that produced by partial throttling of the blood supply to the kidney *a renal hypertension* Ever since Goldblatt s fundamental contribution in 1934[9] numerous modifications of this method have been described, all of which aim at a partial renal ischemia or as in the cellophane wrapped kidney of Irvine Page (1939) [10] a dampening of the pulse wave This renal type of hypertension is not modified by sympathectomy and the nervous system does not seem to be involved in it. Most experimentalists feel that a substance is elaborated in the kidney (angiotonin hypertensin Muñoz) but the striking work of Grollman may indicate that hypertensive disease as it occurs spontaneously in man or as it is induced in the experimental animal is the result of a defect in an incretory function of the kidney [11] It is thus that he produced and maintained experimental hypertension in the bilaterally nephrectomized animal

Human hypertension does develop when the renal artery is obstructed by an atheroma or when a low coarctation of the aorta at the diaphragm produces renal ischemia. We have observed examples of this at the University of Illinois and these will be cited (p 498) Nevertheless the overwhelming majority of essential hypertensive patients shows no obstruction to the renal artery such as the Goldblatt clamp produces To imitate the peripheral arteriolar damage of the nephrosclerotic human Karstens Bedinger Slaughter and I injected sodium morrhuate into one renal artery and later removed the opposite kidney (1931 to 1936) These experiments were never published because of the difficulty of standardizing the procedure Recently however in a master s thesis F L Meine Jr applied this procedure to produce graded renal damage in dogs although the study was directed more towards producing subclinical renal damage [12] Not only nephrosclerotic but also pyelonephritic kidneys show arteriolar damage [13] and the experimental production of diffuse arteriolar disease in the kidney merits further work

Other humoral agents such as cerebrotonin and serotonin may have a considerable role in initiating or maintaining experimental hypertension, but their clinical counterpart is unclear and will not be further discussed here I refer to Irvine Page s summary of the activities of his group and also to his concept of vascular reactivity *i e* an altered response to a vasoactive agent.[14]

The *cortico-adrenal factor in hypertension* has been the object of a study from our clinic [15] The relation of this factor to stress and also to salt

metabolism has stimulated much research activity In a previous article, I summarized the evidence for the significance of this factor in experimental and clinical hypertension [16] Retention and maldistribution of sodium and water occurs in at least these three experimental conditions leading to hypertension hypertension following nephrectomy, hypertension following unilateral perinephritis, and desoxycorticosterone hypertension (Braun-Menendez) [2a] Ledingham feels, however, that this is not true in long-term observations, except relative to the extracellular concentration of sodium in heart muscle, disturbance of water and potassium distribution has not been found.[2a] An important correlation of experimental studies with the clinical state of hypertension is found in observations on salt and water metabolism in patients with essential hypertension compared to normal subjects Particularly, Perera and his associates[2a] have pointed out that desoxycorticosterone has a marked pressor effect in certain hypertensive patients, or in a normotensive person prior to the appearance of hypertension, that the degree of weight loss and diuresis following withdrawal of salt from the diet differs in hypertensive or prehypertensive patients from that observed in normal persons, and that the excretion of sodium after the administration of thiocyanate is increased in the hypertensive patient

Such findings would indicate that essential hypertension could be profitably studied in the so-called prehypertensive stage, not only by the cold pressor test, a test for vascular reactivity, but by some sort of salt tolerance

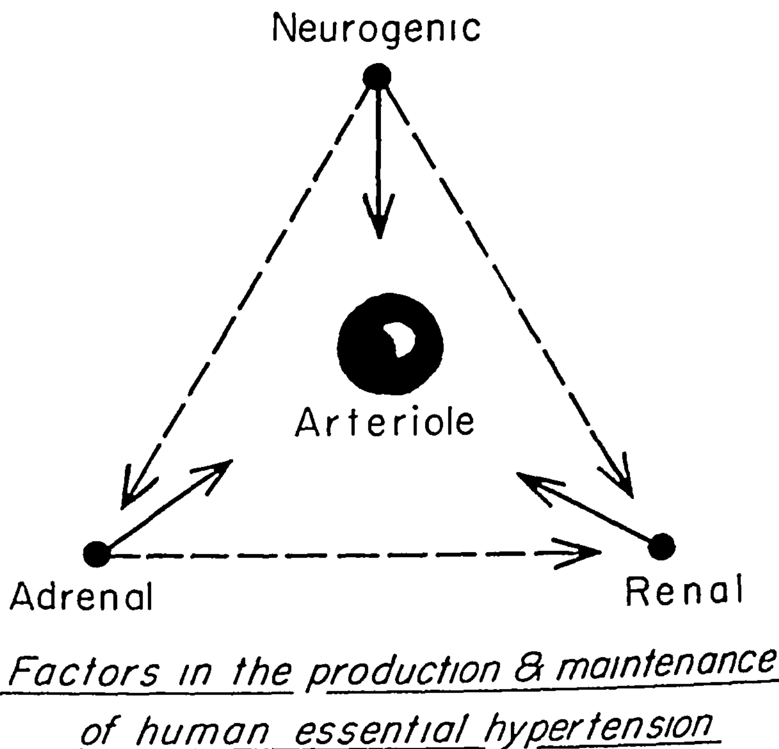


FIG 295 Factors in the production and maintenance of human essential hypertension Each factor increases vascular reactivity, heightens arteriolar tone and influences the other factors Elimination or dampening of one factor intercepts the vicious circle (de Takats, G Limitations of Sympathectomy in Treatment of Diastolic Hypertension J A M A, 148 1382 1952)

or water tolerance test Our own efforts in this direction are discussed on page 491

Finally the syndrome of aldosteronism, suggested by hypertension hypokalemic alkalosis and salt loss in the urine may be present in all hyper tensive patients [17] This syndrome may be secondary however to salt loss

Certainly human hypertension—no matter what its initiating factor—is dependent on a combination of neurogenic renal adrenal and vascular components (fig 295) As will be pointed out in describing the clinical study of such patients these factors can be studied with comparatively simple clinical methods The preponderance of any one of these factors should determine the method of treatment a principle which we emphasized many years ago but which is obviously not followed according to many of the recent reports A vicious circle then develops, in which all these factors participate [1]

CLINICAL FORMS OF HYPERTENSION

Human essential hypertension is not the equivalent of any of the ex perimental hypertensions produced through neurogenic renal or cortico-adrenal mechanisms As pointed out elsewhere [16] these well defined experi mental procedures simply illustrate the mechanisms whereby arteriolar tone can be raised For example an emotional stress originating from the cerebral cortex may operate through all three mechanisms Hypothalamic stimulation will activate the sympathetics mobilize cortico-adrenal secretion through the mediation of the anterior pituitary gland and decrease renal blood flow On the other hand an irreversible arteriolar sclerosis in the kidney may produce all the known characteristics of experimental renal hypertension but a neuro-genic and endocrine factor may be discernible For this reason the clinical measurement of these three mechanisms is the purpose of the hypertensive study and the predominance of one of them may direct treatment

This principle which my associates and I have promulgated in our writings for over a period of 15 years,[16] has been badly ignored by most internists who will either treat hypertension and its complications by reas surance sedatives tranquilizers sodium restriction and ganglionic depres-sants, or will become extremely radical when the process has reached a malignant or an accelerated phase and when surgical help is requested But it is obvious that if hypertension were attacked in its early phase by a more definite approach based on its predominant cause an arrest of the progres sive vascular disease may be accomplished. For this reason, I will now enum erate the clinical measures which help to separate the three mechanisms in the individual patient

The Neurogenic Factor

The cold pressor test of Hines[18] has been the most frequently employed measure of vascular reactivity and if determined in a basal state it reflects

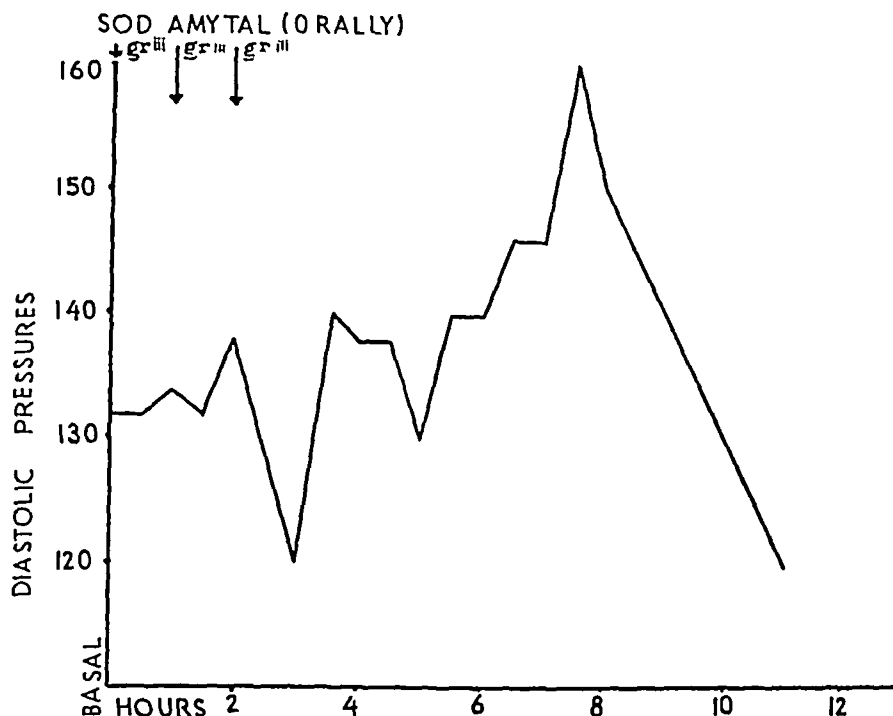


FIG. 296. Sodium Amytal test on a highly tense, middle-aged woman suffering from post-toxemic hypertension She had a violent headache the morning after the test and her diastolic pressure rose to 160 mm Hg Her diastolic floor was 120 mm Hg and she had readily detectable renal damage.

the vascular temperament of a person from birth to grave It is a single, painful stimulus but, as shown by Reiser and Ferris, it may evoke a humoral response that cannot be abolished by a ganglionic depressant [19] A more important objection to the test is that a basal state that is not only physical but also emotional is seldom obtained, and certainly the average response obtained by harrassed interns at the end of the day in a recently hospitalized patient is meaningless. Our group has routinely employed the cold pressor test ever since its original description in the hypertensive work-up (p 528), but we are fully aware of its limitations In the same patient, the response may vary depending upon his emotional tranquility [2f] In my experience, the test is most valuable when used on members of a family of a hypertensive person, recognition of this may have prophylactic value A lack of response with a high diastolic pressure often signifies maximal vasoconstriction

Another test of the neurogenic factor, namely the time-honored administration of 9 gr of Sodium Amytal given in three doses one hour apart during the night, also has its limitations While a statistical correlation between the use of a rapidly acting barbiturate and a ganglionic depressant indicates good agreement,[20] barbiturates have a central effect and depress hypothalamic activity, thus inhibiting the humoral response from the pituitary-cortico-adrenal axis A rebound effect may also occur (fig 296).

It is for this reason that the measurement of the neurogenic factor by a rapidly acting ganglionic depressant, such as tetraethylammonium chloride seems most logical When 6 mg. of this drug per kilogram of body weight are given intravenously the diastolic floor the lowest diastolic pressure obtained within a few minutes after injection, is determined This drug—in our hands—is preferable to Sodium Amytal, to intramuscular hexamethonium (Bistrium) and certainly to Regitine the last drug is primarily adrenolytic but often gives a false positive response (fig 297)

There are other tests of vasomotor activity and from time to time we have made extensive use of the pressor response to carbon dioxide to breath holding, to posture and to exhaling with a closed glottis against measured resistance [21] It needs re-emphasis however, that these tests simply measure vasomotor reactivity which may be increased in a patient with unilateral renal disease with chronic atrophic pyelonephritis or with hypercorticalism Because the carbon dioxide test is such a specific stimulus of the vasomotor center, I reproduce some of our early studies with this agent [22] Figure 298 shows the response of a healthy normal intern to the inhalation of 7 per cent

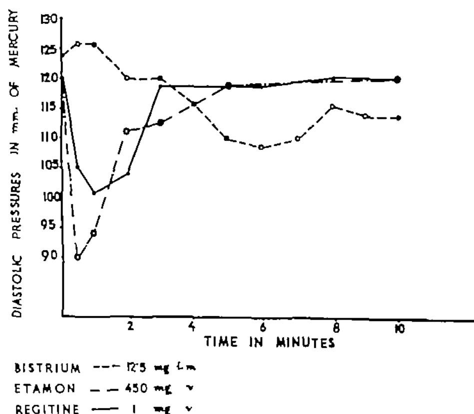


FIG 297 The results of three depressor tests in the same patient, whose Sodium Amytal response is shown in figure 296. Note that intravenous TEAC (Etamon) resulted in a diastolic floor of 90 mm. Hg. The comparison with 12.5 mg. of hexamethonium (Bistrium) is unfair since 25 to 50 mg. doses can be given, but this was the test dose employed by the medical department. Regitine here gave a false positive test in spite of a very small dose. The patient had no pheochromocytoma, at least at its usual locations.

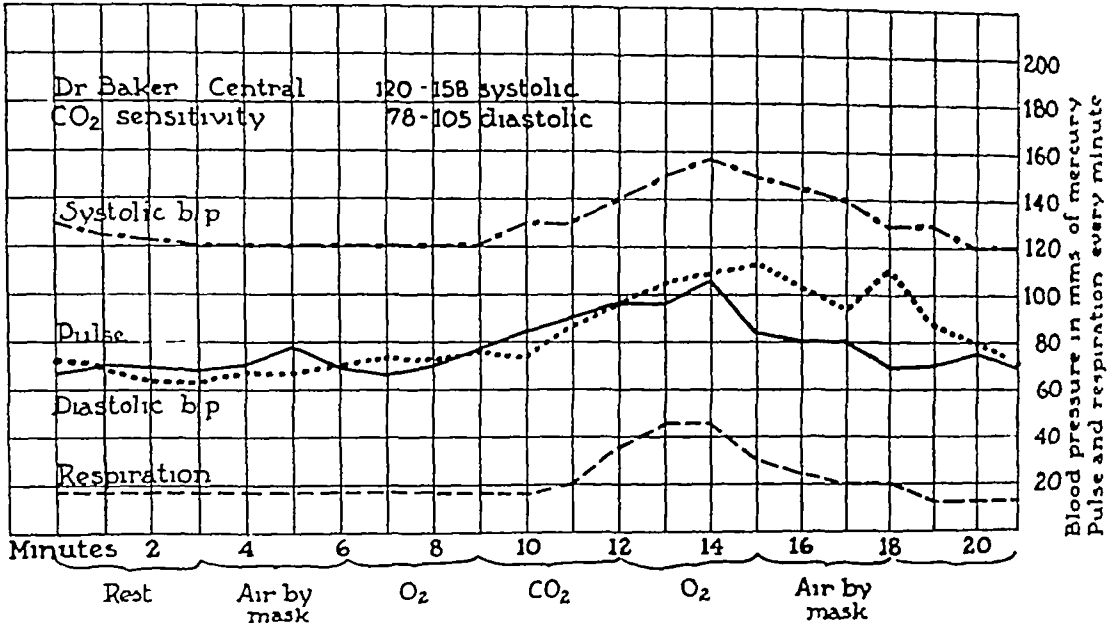


FIG 298 Pressor response to 7 per cent carbon dioxide in a young normotensive man After control periods with rest, air and oxygen by mask (none of which affected the blood pressure, pulse rate or respiration), carbon dioxide was administered for three minutes All lines rose, the peak being reached two minutes after the carbon dioxide has been discontinued This is a normal response (de Takats, G , et al Surgical Approach to Hypertension Arch Surg , *53* 111, 1946)

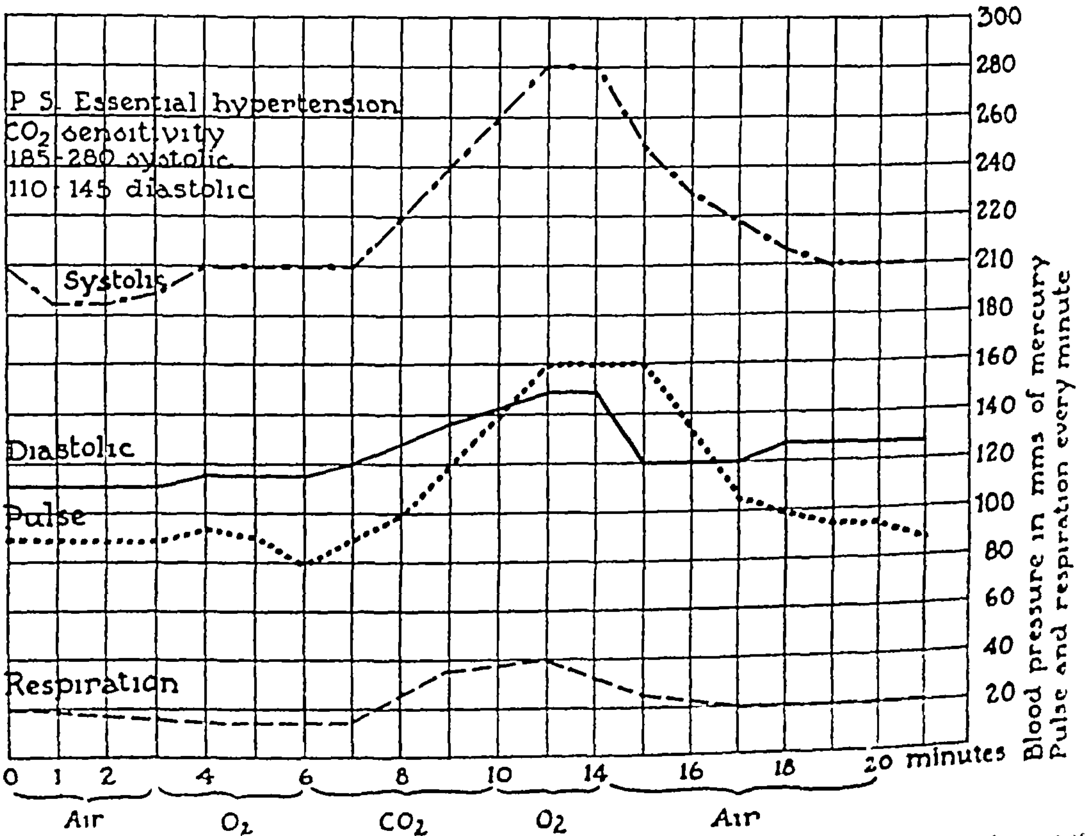


FIG 299 Exaggerated pressor response to 7 per cent carbon dioxide The technique is the same as that used in figure 298 Note the rise of systolic pressure from 210 to 280 mm Hg, and of the diastolic pressure from 110 to 145 mm Hg The pulse rate rose from 80 to 160 a minute This patient showed increased sensitivity to carbon dioxide

carbon dioxide by mask for three minutes Note the rise in systolic and diastolic pressures and the rise of pulse and respiration of a moderate degree and short duration Figure 299 shows an exaggerated response the systolic pressure rising 95 mm Hg and the diastolic pressure rising 35 mm Hg This patient who now has a 12 year follow up after splanchnicectomy has had an excellent result In a third graph (fig 300) the carbon dioxide response of another patient is shown who showed no response to splanchnicectomy He was referred to us from a well known institution in the Middle West with a diagnosis of neurogenic hypertension The renal biopsy taken during *sympathectomy showed* advanced nephrosclerosis In this case the normal response to carbon dioxide had prognostic value Unfortunately because of a change in our Department of Anesthesia at St Luke s Hospital, these studies were not continued particularly in regard to how these patients be have after splanchnicectomy It is most probable, however that Valsalva s experiment (expiration with a closed glottis) is based on a similar mech anism, and Wilkins and his associates have shown us what happens to the Valsalva maneuver after splanchnicectomy[23] (fig 301)

Recent experience with 3 5 per cent carbon dioxide would indicate that this concentration will produce as much cerebral vasodilatation as higher concentrations For this reason a 5 per cent or 3 5 per cent mixture of CO_2 in O_2 seems an adequate physiologic stimulus of the vasomotor center

The Endocrine Factor

A *pheochromocytoma releasing various amounts of epinephrine* and norepinephrine is perhaps the most clear-cut case of endocrine hypertension The clinical syndromes observed in cases of pheochromocytoma will be dis

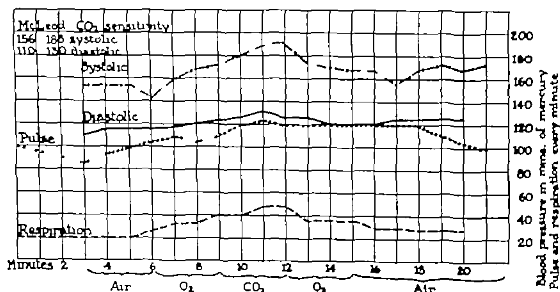


FIG 300 The response of this patient to 7 per cent carbon dioxide is *normal His renal biopsy* showed advanced arteriolar damage and renal function was impaired. He was highly emotional and unstable, but this is primarily a renal hypertension with humoral vasoconstriction. His response to sympathectomy was poor

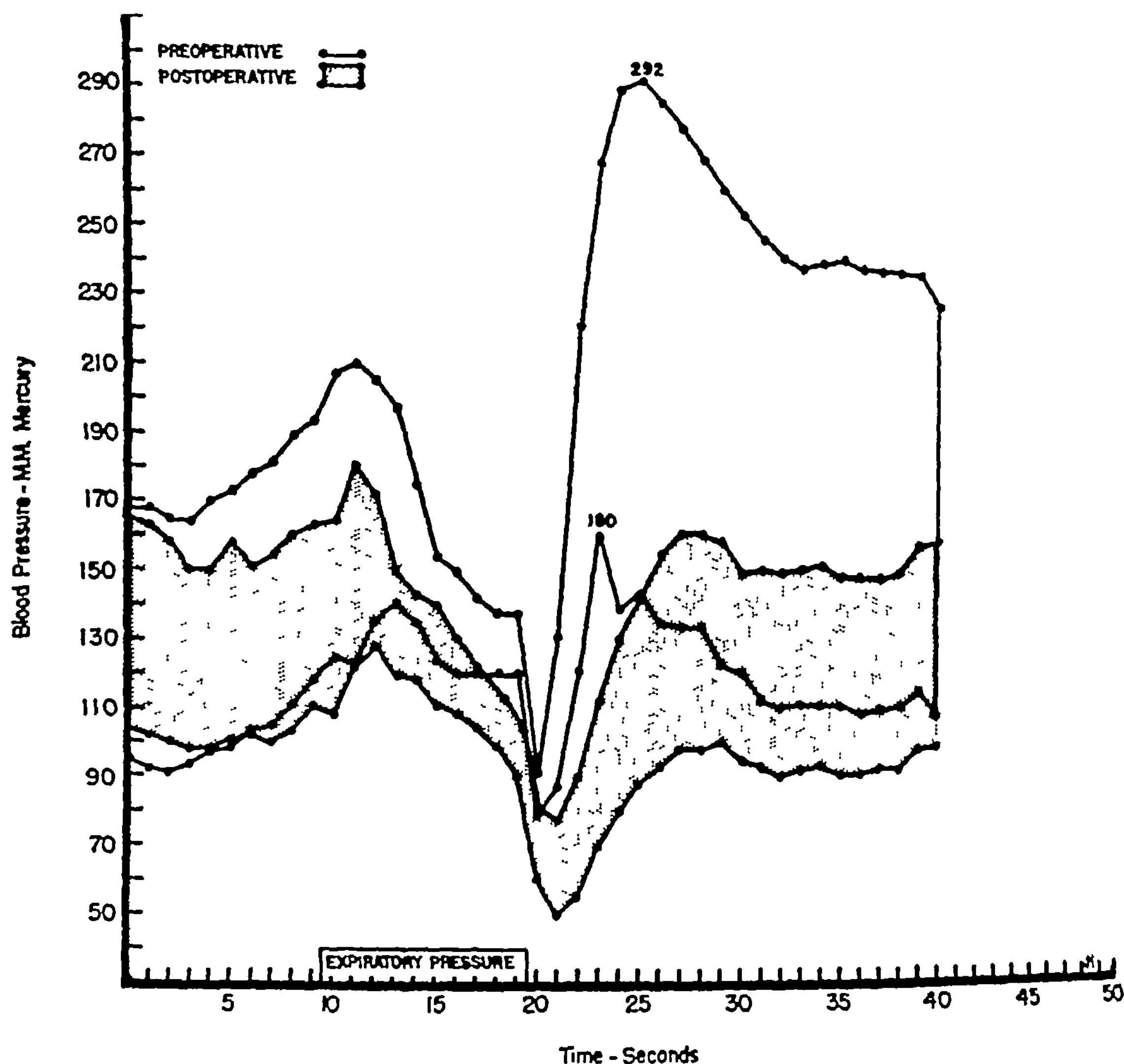


FIG 301. Valsalva experiment before and after bilateral lumbodorsal sympathectomy. Note the tremendous overshoot before the operation, which is eliminated after the operation The basal blood pressures following surgery are essentially the same (Wilkins, R W, et al ' Effects of Various Types of Sympathectomy upon Vasopressor Responses in Hypertensive Patients Surg Gynec and Obst, *87* 661, 1948)

cussed under differential diagnosis (p 502) Here it suffices to point out that only about one third to one fourth of the patients show paroxysmal attacks of hypertension, that many of them show an elevated basal rate and a hyperglycemia, indicating an increased "resting secretion" without pressor effects, and that some of them show a continuous hypertension and even a negative response to benzodioxane, an adrenolytic agent Furthermore, as reported by Goldenberg and Aranow and also by Peart,[2a] hypertension may persist even after removal of the tumor, with a reversal of the previously positive benzodioxane test and with the return to normal of the previously increased pressor amines in the urine This would indicate the point, repeatedly stressed in this chapter, that while the *primary cause* of hypertension may be clear-cut and known, a *secondary hypertension* develops which is not eliminated by the removal of the original cause. We encounter such a phenomenon when a unilateral renal disease causing hypertension is treated by nephrec-

tomy or in cases of coarctation of the aorta even when blood flow is fully restored

In our work not having access to the biologic assay or the chemical determinations of estimating epinephrine and norepinephrine in the urine (Euler Goldenberg, Hamilton and Burn[2c]) we have relied on the responses to two adrenolytic drugs namely piperoxane (benzodioxane) (fig 302) and Regitine *[24] These drugs are of special value when the hypertension is continuous On the other hand, when the problem of producing an attack of paroxysmal hypertension arises the study of an overshoot after the hypotensive effect of histamine or tetraethylammonium chloride (TEAC) has been employed For practical purposes tetraethylammonium chloride is part of our routine hypertensive study and when it produces a posthypotensive hypertension Regitine is used next.[24] The avid almost morbid interest of the house staff in pheochromocytomas has led to the routine use of Regitine especially on the medical services This has resulted in a huge number of unnecessary tests (only about 0 5 per cent of 1 000 surgically treated hypertensive patients have pheochromocytoma) [23] and also in some false positive Regitine tests one of which is shown in figure 297

The tumors are bilateral in at least 10 per cent of the cases and are malignant in about 10 per cent We have seen them located at the aortic bifurcation and believe that the abdominal aorta should always be palpated from the celiac axis to its bifurcation in a search for extra adrenal locations

* The colorimetric method of Sulkovitch (Proc. Soc. Exper Biol. and Med., 59 260 1956) is simple enough for use in a hospital laboratory

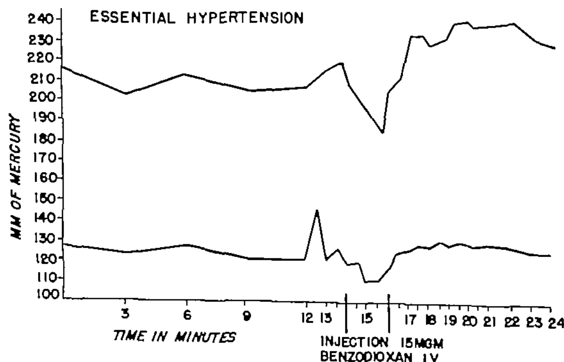


FIG 302. Response of an essential hypertensive patient to benzodioxane A positive response consists of a significant drop in systolic and diastolic blood pressures, returning to control levels in 20 minutes. False negative and positive results may occur This is a negative response.

It is interesting to note that the rate of urinary excretion of epinephrine and especially of norepinephrine is greatly increased in most cases of pheochromocytoma. However, essential hypertensive patients may also show increased norepinephrine excretion in the urine. Von Euler[2c] has studied 500 hypertensive patients with his method of bio-assay and found that 66 per cent of them had a norepinephrine excretion within the normal range, whereas of the remaining 34 per cent only about one half had a significant increase. Even this significant increase (over 60 μg. in 24 hours) is very unimpressive when compared with excretion figures in pheochromocytoma, sometimes to 3,000 μg. a day.

Pheochromocytoma then may be suspected in many cases of chronic continuous hypertension, but then again it is often absent in cases of essential hypertension with attacks of anxiety or with hypoglycemia. The use of aortograms and presacral air injections will be discussed under differential diagnosis (p. 505).

Cortico-adrenal Hyperactivity

Adrenal cortical tumors can be readily divided into two main groups, according to whether they are hormonally active or not.[25] The surgeon who finds a cortical adenoma on exploration of the adrenals in a hypertensive patient must be aware that this mass may not be responsible for the hypertension, since it may be a silent tumor. In the adrenogenital syndrome the production of androgens predominates with a virilizing effect, even though there may be some increased production of cortin. On the other hand, in Cushing's syndrome signs of excessive cortin production predominate, but there may be some evidence of increased androgen or estrin secretion. The full-blown Cushing's syndrome, with a buffalo type of obesity, hirsutism, hypertension, diabetes, osteoporosis and suppression of sexual function, together with laboratory findings leads to the diagnosis of a tumor or hyperplasia of the adrenal cortex. Here we are more interested in the evidence of increased cortico-adrenal function in the essential hypertensive patient, a subject which has been of special interest to our group because splanchnicectomy does not influence this factor and may lead to failure of the operation. In a summary of our studies,[15] I listed some of the simple indirect tests by which this factor was evaluated. While the "oxysteroid" output was determined in an occasional case, patients were studied by tests for insulin tolerance, sugar tolerance and water tolerance. Since the exact technique of these tests will be discussed under methods of study (p. 501), here it is only necessary to outline the considerations which led to the adoption of these simple screening tests. Originally, Fenn, Trump and I,[26] in an effort to distinguish insulin-resistant from insulin-sensitive diabetes, described an insulin tolerance test. The test, of course, is influenced by many factors and figure 303 shows the insulin response in a number of clinical conditions. Note that in the two conditions in which there is pituitary or cortico-adrenal insufficiency, the patients are abnormally sensitive to insulin, behaving like a Houssay dog whose pituitary gland has been removed.

We listed the group of factors which were known to increase sensitivity to insulin among there were the glycotropic anti insulin factor of the pitui tary gland and the anti insulin factor residing in the eosinophil cells of the anterior pituitary gland which requires the presence and functional activity of the adrenal cortex [27] While there was nothing to suggest that this glycotropic factor was hyperactive in essential hypertension, it was known to be present in Cushing s syndrome in which there is notorious insulin resistance and abnormal sugar tolerance When insulin tolerance was determined in 50 consecutive hypertensive patients 31 showed a normal response 4 showed a delayed response and 8 showed no response at all (fig. 304)

Since the insulin resistant group was of interest to us here two addi tional tests were run on these patients and on a number of controls First of these was the intravenous sugar tolerance test of Soskin,[28] the test was selected to eliminate the different rates of absorption of sugar from the gastrointestinal tract Since splanchnicectomy accelerates upper gastrointestinal activity it was felt that the intravenous route would eliminate the different rates of absorption Such intravenous sugar tolerance curves are shown in two patients who were insulin resistant hypertensives The normal curve is shown in comparison (fig. 305) Both other curves belong to insulin resistant hypertensives Case 1 s clinical appearance is shown in figure 306 Her re

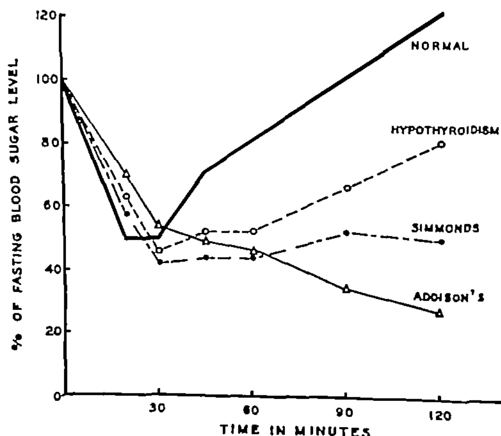


FIG 303 The behavior of the insulin tolerance curve in various clinical conditions. Note that when cortico-adrenal function is diminished either by pituitary or primary adrenal insuf ficiency the patient s blood sugar will remain low in two hours. (Wright, S. Applied Physiology Ninth Edition, Geoffrey Cumberledge, Oxford University Press, London, 1952.)

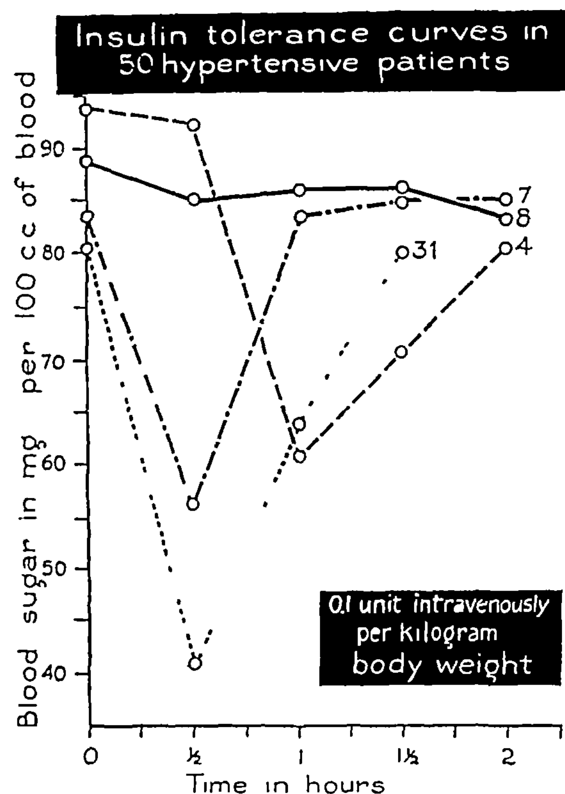


FIG 304 Insulin tolerance curves on 50 consecutive hypertensive patients Note that 31 show a normal response, 4 a delayed response and 8 are resistant to this dose of 0.1 unit per kilogram of body weight, given intravenously (de Takats, G Cortico-adrenal Factor in Hypertension Surgery, *26* 67, 1949)

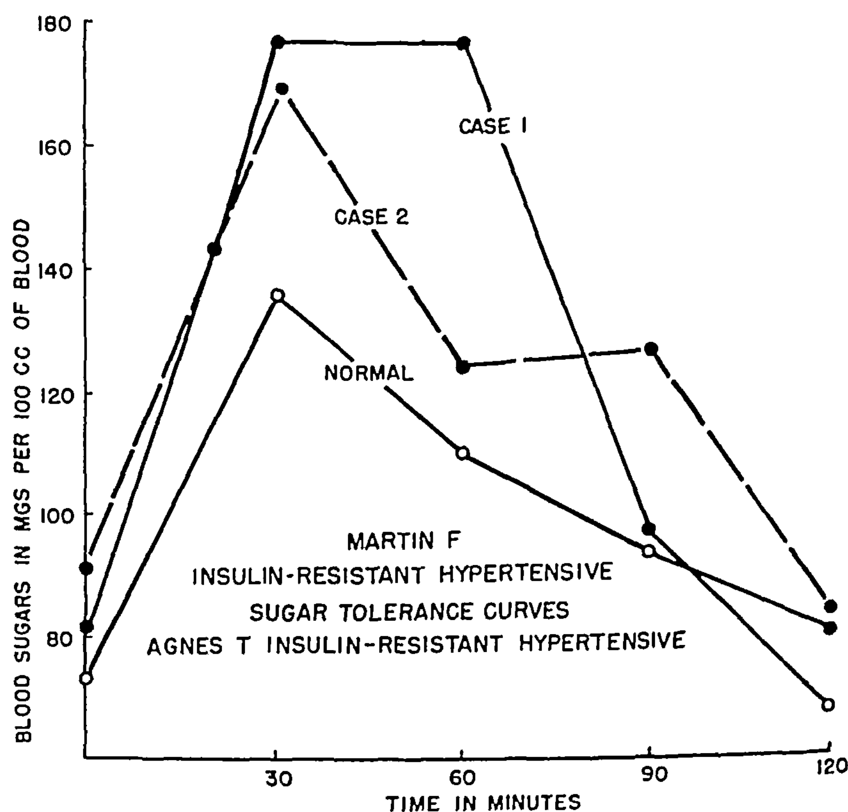


FIG 305 Intravenous sugar tolerance curves The normal curve is shown, determined by Soskin's method [28] Two other curves show a diminished sugar tolerance and belong to insulin resistant patients (de Takats, G Cortico-adrenal Factor in Hypertension Surgery, *26* 67, 1949)

sponse to splanchnicectomy was slow her insulin resistance subsided several months later

The adrenal gland of the patient whose sugar tolerance is shown in figure 305 (case 2) is seen in figure 307 This was a large adenoma of the adrenal cortex intimately adherent to the left adrenal gland which was removed with it

Except for a diminished response to insulin and an abnormal water tolerance, there was nothing to indicate that this patient had anything else but an essential hypertension of 10 years duration with a right hemiplegia which occurred three months before operation Both the insulin and water tolerance became normal after bilateral splanchnicectomy and unilateral adrenalectomy In re-examining his chart a high blood sodium and low blood potassium was found Each hypertensive patient today is studied for the presence of hypokalemic alkalosis, since Conn s description of primary aldosteronism.[29]

So far the tests of insulin tolerance and sugar tolerance were directed toward the sugar hormone of the adrenal cortex which according to Fuller Albright is responsible for the cortico-adrenal activity in Cushing s syndrome [30]

The *salt retaining factor* of the adrenal gland has been of interest in the study of hypertension not only because of a different reaction of some hyper tensive patients to salt deprivation or to salt load [2a] but because of the possi bility that the recently isolated cortical steroid aldosterone is involved in



FIG 306 A.T an insulin-resistant, post eclamptic hypertensive patient with abnormal sugar tolerance curve. She was highly sensitive to TEAC, and had abnormal water tolerance. The left adrenal was slightly firmer and nodu lar She had not a full-blown Cushing's syn drome. Her response was slow but good to splanchnicectomy

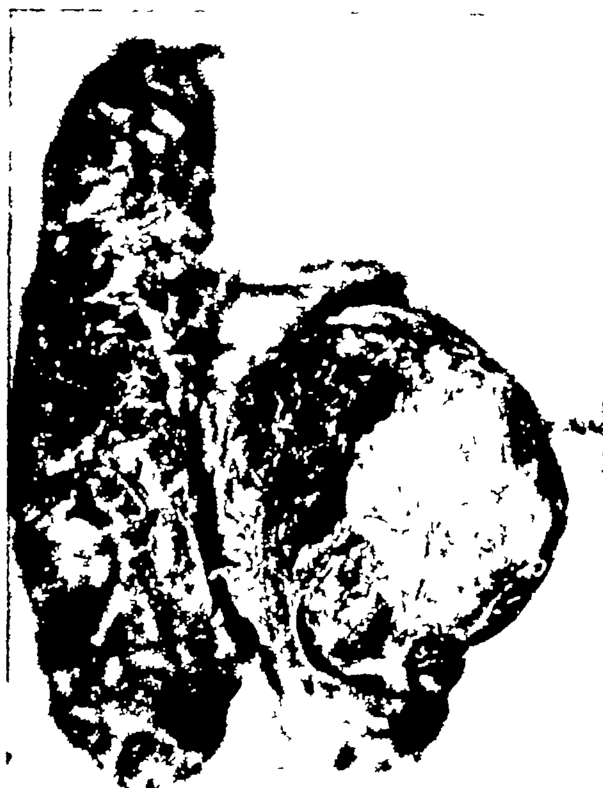


FIG 307. The left adrenal gland with a large cortical adenoma removed during the second stage of splanchnicectomy This was the case of A Y E, who showed a diminished insulin response and an abnormal water tolerance The tests became normal after surgery and the patient had an excellent response to the combined operative procedure (de Takats, G. Cortico-adrenal Factor in Hypertension Surgery, *26* 67, *1949*)

the maintenance of hypertension Thus, Genest and his co-workers have recently indicated that hypertensive patients secrete relatively large amounts of aldosterone [17] The problem of salt metabolism in hypertension has been the subject of many clinical studies[31] and here only our early attempts will be alluded to, because the "water tolerance" as employed by Fowler and me[32] gives a combined picture of adrenal and renal function and because it is simply a small amplification of the concentration dilution test

When a patient on a general hospital diet (about 3 5 Gm of sodium chloride) is made to drink 1,500 cc of water in half an hour, different patterns of water excretion and a change in the specific gravity of the urine will develop, these, of course, are dependent on the salt-retaining factor, the antidiuretic substance in the posterior pituitary gland, and the ability of the kidney to dispose of the water load This test is obviously not specific for adrenal cortical function, but a water-retaining tendency together with a maintenance of high specific gravity in cortico-adrenal hyperactivity is demonstrable. In figure 308, the water tolerance obtained in the case of a cortical adenoma is charted, showing the fixed levels of the specific gravity with a low output of urine in four hours Removal of the adenoma at the second stage resulted in a drop of the specific gravity following the ingestion of water Since in the absence of cortical hyperfunction the test is more a load on renal function, it will be discussed in detail on page 491

The variations of cortico-adrenal hyperactivity from a prehypertensive tendency for salt retention to a full blown Cushing s syndrome are many The interest in evaluating this factor lies in delineating the type of patient, who—if operated upon—needs more than splanchnicectomy Such tests also spare a patient from a bilateral adrenalectomy which in most cases is not indicated in essential hypertension

The Renal Factor

The functional state of the kidney is of dominant interest in the treat ment of hypertension because whether the renal damage is due to a primary insult or whether it is the result of repeated neurogenic or endocrine stresses it is the least likely to be reversible The fixed vascular damage is arteriolar sclerosis and is less frequently due to obstruction of the main renal artery as in a Goldblatt kidney

The inulin D odrast clearance so widely used in clinical research is not a practical test we have felt however, that our modified concentration dilution (water tolerance[34]) test a 15 minute phenolsulfonphthalein (P S P) test and a urea clearance give sufficient information regarding the state of

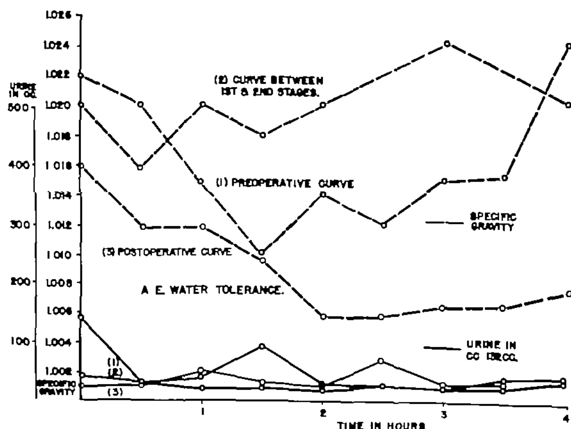


FIG. 308 Specific gravity and urinary output after the ingestion of 1,500 cc. of water in A.E. on whom right splanchnicectomy was done in the first stage and left splanchnicectomy and adrenalectomy done in the second stage. Note the fixed specific gravities before and after the first stage. In fact, the second curve is even more fixed than the first, perhaps as the result of increased postoperative activity of the adenoma. The third curve shows a drop of specific gra vity but this is still an abnormal curve.

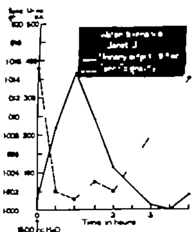
the kidneys in the individual patient, although these tests are obviously crude

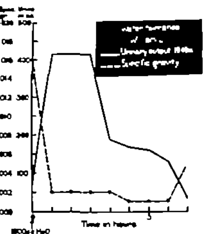
The water tolerance test, as we have demonstrated, is influenced by desoxycorticosterone and other salt-retaining factors, by the antidiuretic hormone and also by hepatic disease for which it was first employed Nevertheless, with the exception of detecting marked cortico-adrenal activity, it primarily measures the ability of the kidney to adapt itself to a sudden stress, such as an intake of 1,500 cc. of water Since this amount, which is to be ingested in a half hour, nauseates many patients or makes them vomit, the water load was recently decreased to 1,000 cc Figure 309 shows the various patterns of urinary excretion and of specific gravity which develop after this physiologic stress Note that concentrating ability is maintained in five of the six patterns The ability to reconcentrate urine in the first hours is present only in Janet J , whose pattern is identical with that obtained in young, normotensive individuals The kidney tries to compensate for the decreased concentrating ability with an increased urinary output It seemed to us that certain patterns signify irreversible renal damage or the presence of such factors, which sympathectomy cannot influence In correlating the water tolerance of patients with the results obtained by dorsolumbar sympathectomy, it is to be noted that failures increase with diminishing response to the water load When the water tolerance was correlated with a 15 minute P S.P test, it seemed to decrease as the 15 minute P S P excretion diminished [16]

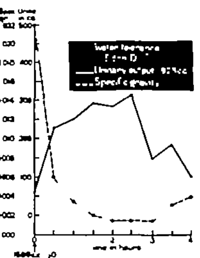
Since the water tolerance may well be influenced by increased sodium and water retention or by emotional stress, two additional renal function tests are employed, namely a 15 minute P S P test and the urea clearance test While there are limitations to both of these tests, the combined result of the three, *i e* , of the water tolerance, the 15 minute P.S.P test and the urea clearance test, gives an adequate estimate of the severity of renal damage

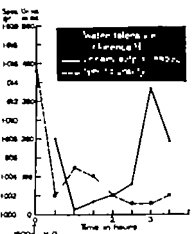
As mentioned before, glomerular filtration rate and total renal blood flow have been employed on our service at the Research and Educational Hospitals of the University of Illinois with the help of the late R W Keeton, with whom many of these cases have been studied These tests, however, are so sensitive that they may be influenced by the emotional state of the patient Wolf and his associates[33] have published convincing data to show that in both normotensive and hypertensive patients the kidney adjusts itself by means of vasoconstriction to the rise of systemic blood pressure, thus preventing an increase in blood flow through it They also found that renal vascular resistance becomes much higher and persists much longer in a hypertensive than in a normotensive individual, and herein might lie the origin of renal vascular disease after repeated chronic bouts of stress

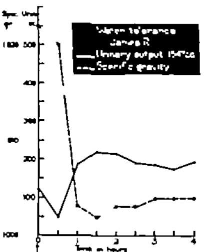
The experiments of Trueta and his co-workers,[34] showing ischemia of the renal cortex on stimulation of the sciatic nerve, are good examples of the labile vascular state of the kidney (fig 310) While the renal shunt mechanism which diverts blood from the vascular bed of the cortex is not accepted











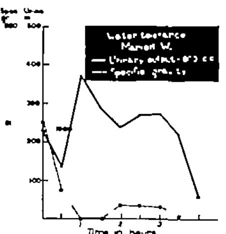


FIG 309 The water tolerance of hypertensive patients, showing six patterns of decreasing ability of the kidneys to handle 1,500 cc. of water Starting from the top row left to the bottom row right, urinary excretion is delayed and specific gravity of the urine remains low instead of returning to a level obtained by the concentration test The patients showing the top three patterns showed excellent results from sympathectomy Those exhibiting the lower row patterns have either been refused operation or were surgical failures. (For more details see de Takats, G Causes of Failure in Surgical Treatment of Hypertension Angiology *1* 457 1950)



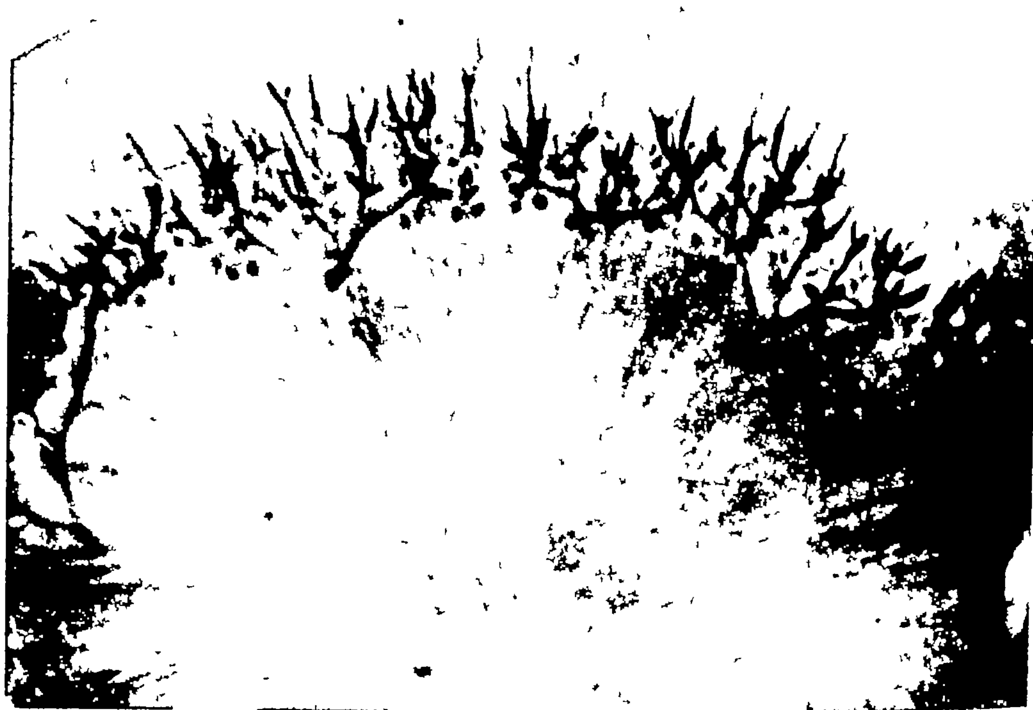


FIG 310 Radiomicrographs of rabbit kidneys (a) The contrast medium fills the interlobular arteries to their termination The vasa recta are not seen (b) Following a tourniquet on the hind limb as a potent sensory stimulus, the cortex of the kidney becomes pale, and the terminal segments of the interlobular arteries are constricted The proximal segments of these arteries are well filled and so are the vasa recta and many juxtamedullary glomeruli (Trueta, et al Studies of the Renal Circulation Charles C Thomas, Springfield, Ill, 1947)

by many workers and while massive terminal vasoconstriction could also explain such a phenomenon, there can be no doubt about a functional change of nervous origin in the blood flow, which can be abolished by sympathectomy

The clinically employed "crude" tests, however, give a more clear-cut

indication of fixed renal damage The correlation of such renal function tests with histologic findings obtained by biopsy have been repeatedly attempted Castleman and Smithwick[35] found that in half of their patients the evidence of renal arteriolar disease was absent, minimal or mild, indicating that renal arteriolar disease was not a necessary precursor to the hypertensive state but, on the contrary develops along with or subsequent to hypertension On the other hand, an excellent correlation of renal clearance studies and renal biopsies was shown in another study on Smithwick s material [36]

A detailed report of our observations on renal biopsies was given in 1946 [22] Between 1939 and 1945, 65 renal biopsies were studied by the pathol ogists associated with our group * Since this last study renal biopsies have been consistently taken during the first stage of a bilateral lumbodorsal sympathectomy We have come to regard renal biopsy which in recent years has been done on a large scale as a punch biopsy by Kark and his associates from our institution [37] as a help in determining the nature of renal disease and also as an index of prognosis It certainly tells more than muscle biopsies do these have been correlated by Heyer and Keeton[38] from our institution with renal biopsies They concluded that regardless of the origin of the hypertension the histologic changes were the same hypertrophy of the muscular layer constriction of the lumen and an alteration of the lumen to wall ratio as first emphasized and graded by Kernohan Anderson and

* Drs. R. M Jaffe, E. F Hirsch and S R. Rosenthal Drs. Granville R Bennett, and R. J Jensik offered valuable counsel.

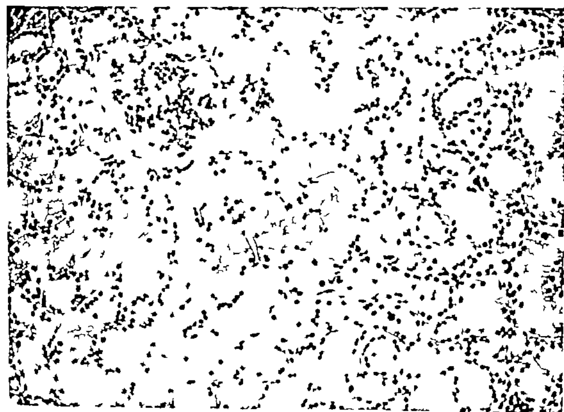


FIG 311 Vascular changes, group 1 An essentially normal glomerulus and tubules are seen. One arteriole above the tuft shows beginning hyalinization.

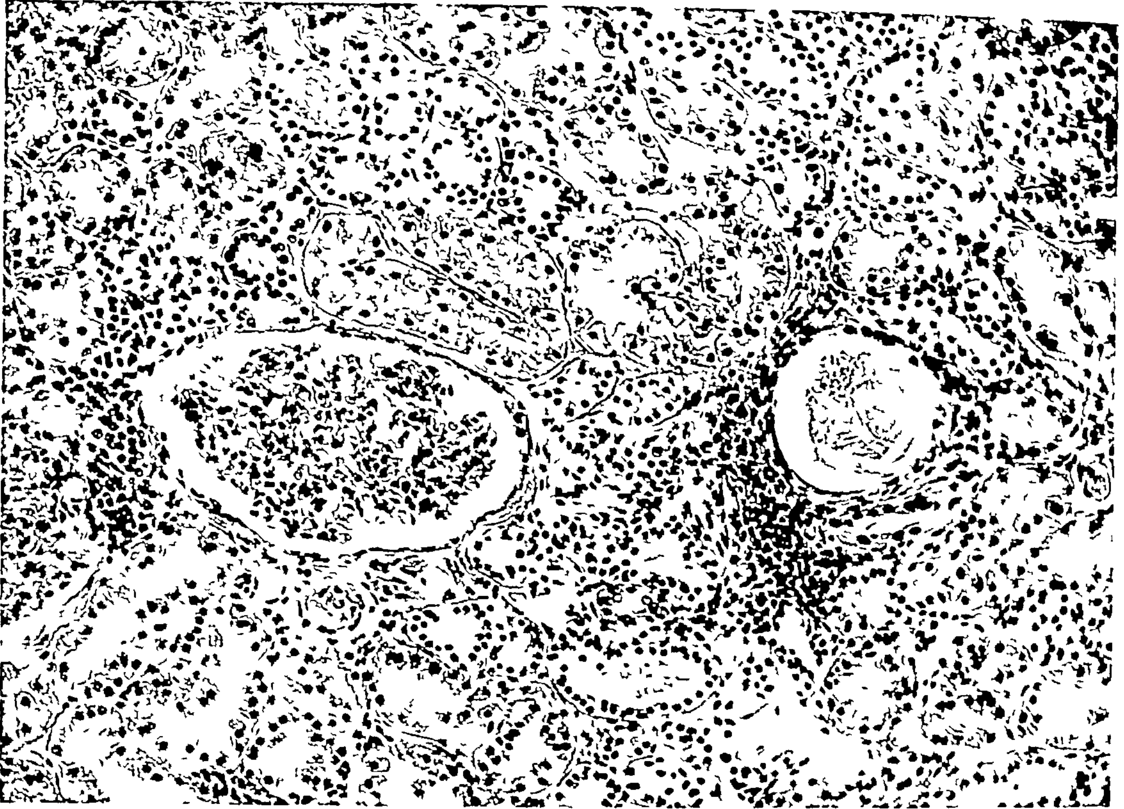


FIG 312 Vascular changes, group 2 Two arterioles below the glomerulus demonstrate more pronounced hyalinization There is some scarring and round cell infiltration of interstitial tissues

Keith [39] Hypertension could thus be graded and clinically correlated with specimens of muscle Our experience has been, however, that the method of fixation and the site of the biopsy specimen may influence the lumen to wall ratio, and unless careful micrometer measurements are made by the same person, nothing more than luminal constriction can be diagnosed This is permanent and will not improve after a sympathectomy, even though the pressure returns to normal

The renal biopsy, on the other hand, may reveal chronic pyelonephritis when nephrosclerosis is diagnosed and may show various stages of glomerular nephritis, streptococcus nephritis and lesions accompanying collagen disease, all of which may be masked under the clinical diagnosis of essential hypertension

Biopsy specimens classified as belonging to group 1 demonstrate beginning subintimal hyalinization of the scattered arterioles Occasionally, vessels over 100 μ in diameter reveal a slight thickening of the media There may be an occasional, narrow, wedge-shaped area of fibrosis and a few glomeruli may be scarred (fig 311)

In group 2, the changes are more advanced and the vessels are immediately more conspicuous The subintimal hyalinization is prominent in the involved arterioles and is present as a thick complete collar of pink-stained material Furthermore, in vessels over 100 μ, beginning reduplication of the internal elastic membrane is apparent Usually more glomeruli are hyalinized and focal infiltration of round cells is seen (fig 312)

In biopsy specimens classified as belonging to group 3 all vessels are involved The arterioles contain hyalin rings of varying thickness and in several of the larger arterioles and smaller arteries reduplication of the elastica is prominent Distinct narrowing of the lumina is obvious and sizable, wedge shaped areas of fibrosis and cellular infiltration are present (fig 313)

In the fourth group every vessel is involved and the narrowing of the lumina has advanced to almost complete obliteration in many instances Hyalin can be seen in many of the arterioles but others show pronounced intimal proliferation and reduplication of the elastica with narrowing of the orifices to mere slits Necrosis of the afferent arteriole and glomerulus may be observed and this of course is characteristic of the malignant phase The interstitial scarring is not so pronounced as in the previous group suggesting a more rapid tempo of the arterial and arteriolar changes (fig. 314)

The results of these biopsies were published in 1946 [2-] They follow the terminology of Moritz and Oldt in describing subintimal hyalinization medial hypertrophy and endothelial hyperplasia with reduplication of the intimal elastic membrane as employed by Castleman and Smithwick [35] This question immediately arises what information can the renal biopsy furnish toward a better understanding of the individual case and does it have more prognostic value than the clinical study including muscle biopsy? We raised these questions 12 years ago[22] and today they seem even more perti

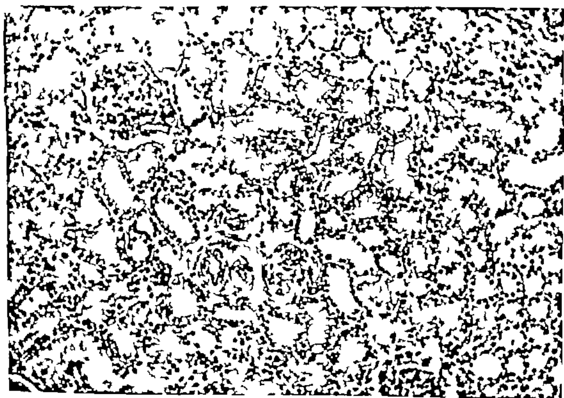


FIG 313 Vascular changes, group 3 The arterioles are more prominent their walls are hypertrophic and hyalinized. A glomerulus in the left upper quadrant is shrunken.

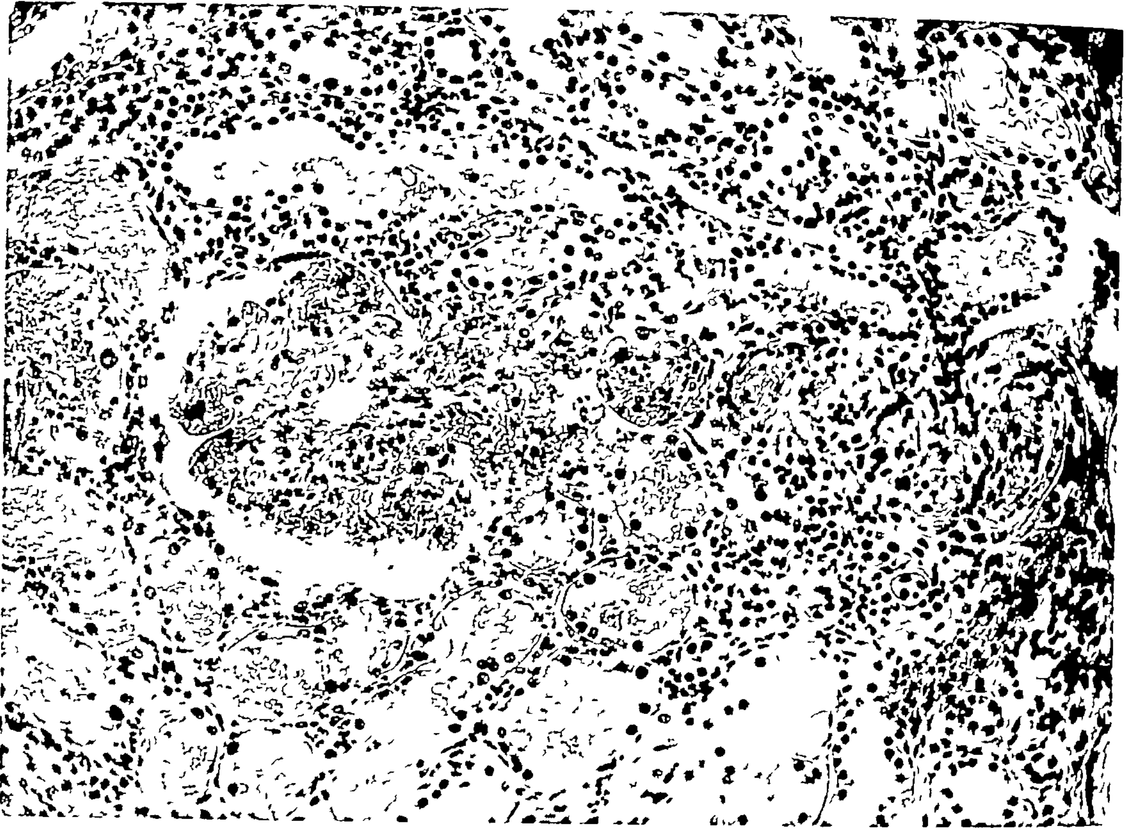


FIG 314 Vascular changes, group 4 This specimen is from a patient in a malignant phase of hypertension There is necrosis of the tortuous afferent arteriole and of the glomerular tuft

nent because of the use of percutaneous renal biopsies, whose hazard, while small, is definite.

It is true that healed nephritis and pyelonephritis may masquerade as essential hypertension, furthermore, other diffuse vascular disease, such as lupus erythematosus, thromboangiitis obliterans and panarteritis nodosa, may be recognized in the kidney when clinically little more than sustained hypertension and diffuse vascular disease exists (fig 315) Actually, however, such renal sections, like the peripheral arterial biopsies, are usually studied in a late stage, and while the differential diagnostic criteria between pyelonephritis and nephrosclerosis have been clearly described by Weiss and Parker,[40] in later stages the differentiation between inflammatory and degenerative lesions may be difficult if not impossible

The question of whether or not a renal biopsy gives more prognostic information than the clinical study with muscle biopsy must be answered in the affirmative I have already alluded to the varying amount of emotional stress during a hypertensive study and also to the fact that muscle biopsies nowhere run a parallel course with renal biopsies, but this does not mean that one would wish for a renal biopsy unless the kidney was in the surgical field, and it need not be obtained by a needle puncture The reaction of some patients to this procedure is such that the procedure, at least at present, can only by justified for clinical investigative purposes, but seldom to direct therapy and offer prognosis

Aortograms seem important to detect (1) an adrenal tumor especially if combined with presacral insufflation of air (fig. 316) but this would be done only if clinical or laboratory findings point toward this possibility and (2) to visualize a segmental obstruction of the renal artery leading to unilateral or even bilateral renal ischemia We were stimulated to look for such lesions after the provocative article of Blackman appeared in 1939 [41] he found in postmortem studies that arteriosclerotic plaques projected into the renal arteries in 86 per cent and produced marked stenosis in 25 per cent of 50 cases of essential hypertension In spite of exploring the renal arteries in every case of lumbodorsal sympathectomy for hypertension and performing aortograms on a series of hypertensive patients since 1950 we have encountered only a single case of renal arterial obstruction They do occur however although the finding of an obstructive lesion of the main renal artery need not necessarily be the cause of hypertension When however the hypertension is of sudden onset and is accompanied by unilateral abdominal pain aortography is certainly indicated Howard and his associates[42] recently described six cases of an acute onset with severe hypertension all of which were due to vascular lesions in major branches of the renal artery due to thrombosis and embolism and leading to renal infarcts Nephrectomy



FIG 315 A number of glomeruli show shrinking and fibrosis, some show thickening of Bowman's capsule. The small arteries and arterioles show marked thickening of their walls, and sometimes are fully obliterated. The tubules in the cortex show alternate distention and collapse Interstitial tissue is increased. This patient's clinical course was that of segmental inflammatory arterial disease in the extremities with hypertension later there were some cerebral vascular changes. The clinical diagnosis was that of thromboangiitis obliterans with visceral manifestations. The patient died of generalized arteriolar sclerosis superimposed on his inflammatory vascular disease.



FIG 316 Combination of aortogram and presacral insufflation of air In this hypertensive patient, a suspicion of pheochromocytoma was entertained Note the visualization of the two renal arteries and of the branches of the celiac axis The rete of vessels to the right adrenal gland is visible No adrenal tumors are seen The metal clips are the result of a dorsolumbar sympathectomy Residual barium is in the left half of the colon This was a case of "essential" hypertension with no renal artery involvement or adrenal tumor

cured the hypertension On the other hand, Fishberg[43] described five cases of embolic obstruction of a renal artery, none of which produced a lasting rise in blood pressure

More recently, Paul DeCamp has given a complete description of renal artery stenosis associated with hypertension [44] Both his group at the Ochsner Clinic and that of Poutasse and Humphries at the Cleveland Clinic[45] published cured cases of hypertension either by nephrectomy or by restoring the continuity of the stenosed or blocked renal artery The suspected clinical diagnosis may be confirmed by aortography

In addition to looking for unilateral renal disease due to pyelonephritis or embolic infarcts, ligation of an aberrant renal artery or pressure on the renal artery from invasive retroperitoneal tumors needs to be considered Renal ptosis, especially the small pelvic kidney, created a considerable stir following the report of McCann and Romansky (1940) (cited in 46)

The presence of the renal factor in "essential hypertension" can be suspected or corroborated by (1) a good history of glomerulonephritis or

pyelonephritis (often vague or missing) (2) urinary cultures (often negative in healed or quiescent lesions) (3) an intravenous pyelogram which we believe is a routine part of the hypertensive work up and which again does not always show the deformed calices (4) an aortogram in cases suggestive of unilateral renal disease and (5) a renal biopsy mostly during exploration and not as a blind procedure

Occasionally other renal diseases such as a polycystic kidney periarteritis nodosa and lupus erythematosus come to light The point to be emphasized is that even though the initial hypertension may have been fully renal chronic sustained hypertension which is primarily renal in origin has other factors in operation, such as neurogenic and endocrine not to mention the effect of the hypertension itself on the arteriolar tree Our group has registered cases of renal hypertension which have done remarkably well following splanchnicectomy This was true of postnephritic and post toxemic hypertension Fowler and I[46] found that removal of a small hypoplastic or atrophic kidney did not necessarily relieve hypertension until a bilateral splanchnicectomy was done In many children nephrectomy is delayed until arteriolar sclerosis of the opposite hypertrophic kidney produces irreversible malignant and rapidly fatal hypertension Such case reports were published from our clinic in detail

Two patients in our series had well documented renal trauma and were treated not by nephrectomy but by bilateral splanchnicectomy Two and three year follow ups revealed normal blood pressures

Best results were obtained in the *post toxemic hypertensive group* The renal lesion secondary to toxemia is an example of a state in which endogenous factors probably originating in the placenta produce structural arteriolar changes in the kidney these in turn by producing renal ischemia, liberate pressor substances which cause hypertension [47] As pointed out succinctly by Pickering,[2c] the exotic concepts as it were the hot house plants of specialism such as the low reserve kidney and occult nephritis are on the way out Obviously two conditions have to be distinctly separated (1) the pre-eclamptic and eclamptic toxemia characterized by the appearance of hypertension edema and proteinuria, never before the twentieth and nearly always after the twenty fourth week of pregnancy or (2) if the blood pressure is elevated before the twentieth week of pregnancy the case becomes one of hypertension preceding or independent of pregnancy and if proteinuria supervenes it becomes one of toxemia complicating hypertension Our cardiovascular service in close cooperation with the obstetricians* have studied a number of cases during and after pregnancy and also following splanchnicectomy after the patients became pregnant again A few points need repeated emphasis the fall of pressure after delivery is so reassuring to the physician and patient alike that they do not fear that another pregnancy may again elevate the blood pressure Only since the studies of Herrick and Tillman, Browne and Dodds, and Chesley Somers and Vann (cited in 2c) has it be

* The late Dr H O Jones, Dr Eugene Edwards and Dr Robert Beebe.

come clear that between 30 to 50 per cent of the women exhibit a continuous, progressive hypertension later, and of those whose pressure is normal when not pregnant, there is a high rate of recurrent toxemia. They all agree that the *duration of toxemia*, not necessarily the height of blood pressure, determines the rate of recurrence and also the incidence of the malignant phase.

I have gone into more detail in describing this type of hypertension, because our best results following splanchnicectomy have been in this group. The reasons for this are: (1) these patients belong to a younger age group with little arteriosclerosis, (2) women always have a better prognosis than men, and, chiefly, (3) the original insult (toxemia), if not allowed to persist and not allowed to recur with another pregnancy, does not operate continuously. Splanchnicectomy seems to permit another pregnancy with little and only late rise in blood pressure, provided renal function is adequate. We have published case reports with Fowler[46] and are following a small series with Beebe after splanchnicectomy and through their second pregnancy.[48]

Post-toxemic hypertension is thus of great theoretical interest. It is the result of a comparatively short toxic damage to the vascular endothelium, leading to enlarged swollen glomeruli, periportal hemorrhagic lesions in the liver and infarcts in the placenta due to acute atherosis of the decidual vessels.[49] All this is reversible, and the renal lesion producing hypertension may be adequately counteracted by splanchnicectomy. Here is then a non-neurogenic, renal type of hypertension, which, provided the organic changes have not gone too far, responds to sympathectomy better than does a "neurogenic" lesion.[46]

Equally significant is our experience with hypertension in *periarteritis nodosa*. We[50] reported a case of periarteritis nodosa following thiouracil therapy, in which a marked, progressive hypertension was eliminated by early sympathectomy, with no recurrence for many years (10 years at this writing). Here is a disease in which the basic lesion is a wide-spread fibrinoid necrosis of the arterial wall, affecting, among other organs, the kidney and producing necrosis of the glomerular tufts and a periglomerular inflammatory reaction. When hypertension develops, it seems to be the consequence of a healed periarteritis nodosa of the renal arteries, but this hypertension, provided it is not allowed to persist, responds well to sympathectomy. Only one other case like this has been reported and was equally successful,[51] indicating that a renal lesion has initiated a course of events (a vicious circle) which can be arrested by sympathectomy.

When one realizes that of 55 cases of periarteritis nodosa, 11 terminated with a malignant phase of hypertension (G. A. Rose cited by Pickering in 2c), it can be seen that splanchnicectomy is an important weapon against such a complication. This indication—as far as I can tell—is ignored both by internists and surgeons and needs more stress than we have given it in the past.

One experience with bilateral polycystic kidneys and one with a coarctation of the aorta, seen before Gross' report on direct surgery of the lesion,[52] convinced me that even such lesions, when treated with splanchnicectomy,

can obtain a long lasting reduction of blood pressure primarily due to a nonmechanical factor It has been repeatedly stressed that hypertension in coarctation of the aorta is not simply an increase in the peripheral resistance at the stricture and in the collaterals [53] When the coarctation is low at the diaphragm severe hypertension develops which cannot be always eliminated by a bypass (Julian and Dye from the University of Illinois College of Medicine) The hypertension then can be of renal origin or due to a reflex vasoconstriction The fact remains that after a satisfactory excision and repair of the coarctation or after a graft placed into the retracting two segments residual hypertension may present itself in spite of no narrowing at the site of anastomosis [c]

METHODS OF STUDY

A four day hypertensive work up was inaugurated on our service about 20 years ago The outline of such a study has been repeatedly modified depending on the prevailing interest of our group in certain aspects of the problem Gradually this study has been limited to simple clinical tests minimizing duplication unnecessary delay or expense to the patient at a private institution On the teaching service more elaborate or time-consuming tests were frequently done but no additional information than obtained from the simpler procedures has been forthcoming. Since our medical colleagues were pleased with and also utilized this form of study over 800 patients have been put through this type of observation

Mimeographed sheets are distributed to the residents and floor nurses (appendix I p 528) The consulting ophthalmologist and internist are asked to see the patient the first day and then again after completion of the study

Early in our experience the resident in neuropsychiatry was asked to interview each patient especially if an operation was contemplated. For the last 10 years, however a psychiatric social worker has talked to the hypertensive patient during the whole period of observation Over 100 such reports are now in our files * It is explained to the patient that such an interview is just as important for a thorough evaluation of the case as a renal function test or an electrocardiogram Occasionally a rapid preoperative and postoperative therapy as outlined by Alexander and French[34] may tide them over this stressful period The background life experience and attitudes of hypertensive patients warrant serious consideration [2f]

The questions to be answered by such a study are as follows (1) what is the participation of the neurogenic cortico-adrenal and renal elements in a given case of essential hypertension (2) can a definite surgically removable cause of hypertension be found, such as an unilateral renal disease pheochromocytoma, cortico-adrenal adenoma, hyperplasia, coarctation of the aorta or renal artery stenosis (3) if the hypertension is essential and demonstrably progressive, does the existing renal damage prohibit any surgi

*A sample of such a study is found at the end of this chapter page 532.

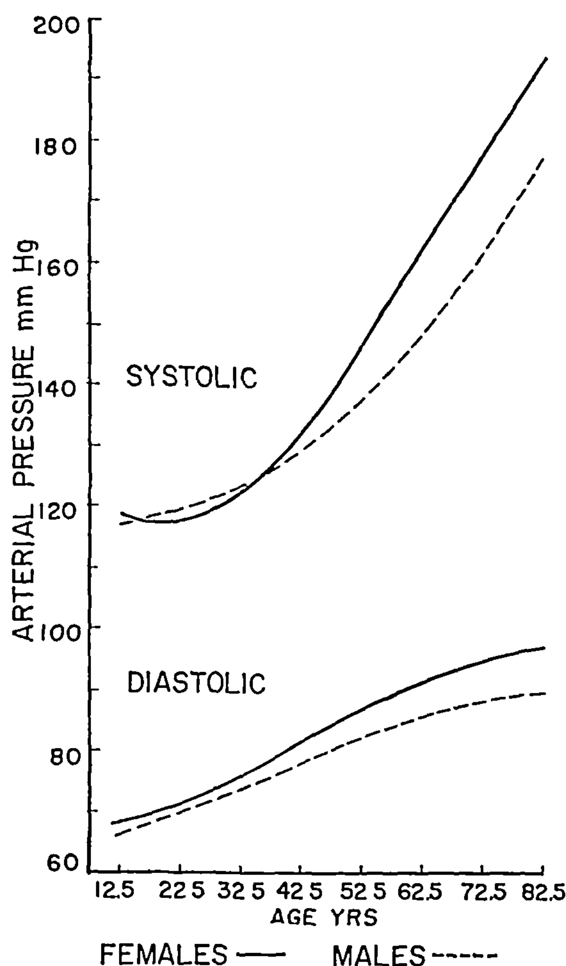


FIG 317 Systolic and diastolic pressures for females and males for each five year age group of the population sample, together with the fitted curves (Redrawn from Hamilton, Pickering, Roberts and Sowry Aetiology of Essential Hypertension Clin Sc, *13* 11, 1954)

cal relief, (4) is the emotional state of the patient such that an operative procedure, while resulting in a lowering of blood pressure, may do him more harm than good, or should he have preoperative psychotherapy and, (5) are his symptoms due to a hypertensive neurosis in analogy with the cardiac neurosis superimposed on an organic cardiac lesion As will be discussed under treatment (p. 511), strict observation of the limits of operability have been responsible for a comparatively small percentage of failures.[16]

DIFFERENTIAL DIAGNOSIS

As a result of a hypertensive study, certain well defined groups can be distinguished and separated from 90 per cent of the patients grouped under the generic term of "essential hypertension"

Systolic Hypertension

Systolic hypertension, which, of course, is not the object of this present discussion, may occur in conditions which increase the stroke volume of the heart, such as in bradycardia due to heart block, aortic regurgitation, arterio-venous fistula, patent ductus arteriosus, thyrotoxicosis, fever and other conditions which increase pulse pressure and which can be reproduced by the effect of intravenous epinephrine. Most importantly, high pulse pressure

occurs in degenerative changes of the aorta and its main branches and in most cases of coarctation The increase in pulse pressure with age in the general population is shown in figure 317 The sharp rise in systolic against the slow rise of the diastolic pressures is striking In many clinics such pa tients have received intensive dietary medical or even surgical therapy whereas the systolic rise—in the absence of a mechanical obstruction such as a coarctation—is probably a compensatory and desirable phenomenon and should not be tampered with

Only in paroxysmal attacks of pheochromocytoma may a huge systolic rise and little if any diastolic rise be observed but these are the rare pheochromocytomas in which the effect of epinephrine predominates over that of norepinephrine thus creating high peripheral resistance Generally speaking, systolic hypertension, omitting the two exceptions of coarctation and pheochromocytoma is not a surgical disease and need not and cannot be lowered

Coarctation of the Aorta

This should be always looked for during the initial examination by com paring the radial and femoral pulses In several patients referred to us for

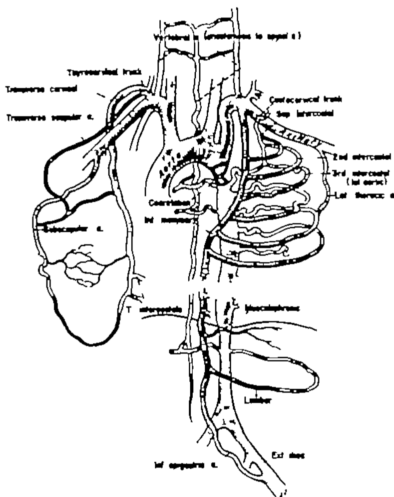


FIG 318 Collateral circulation in coarctation of the aorta at the usual level. (Allen, E., Barker N and Hines, E. A., Jr Peripheral Vascular Diseases. Ed. 2. W B. Saunders Co)

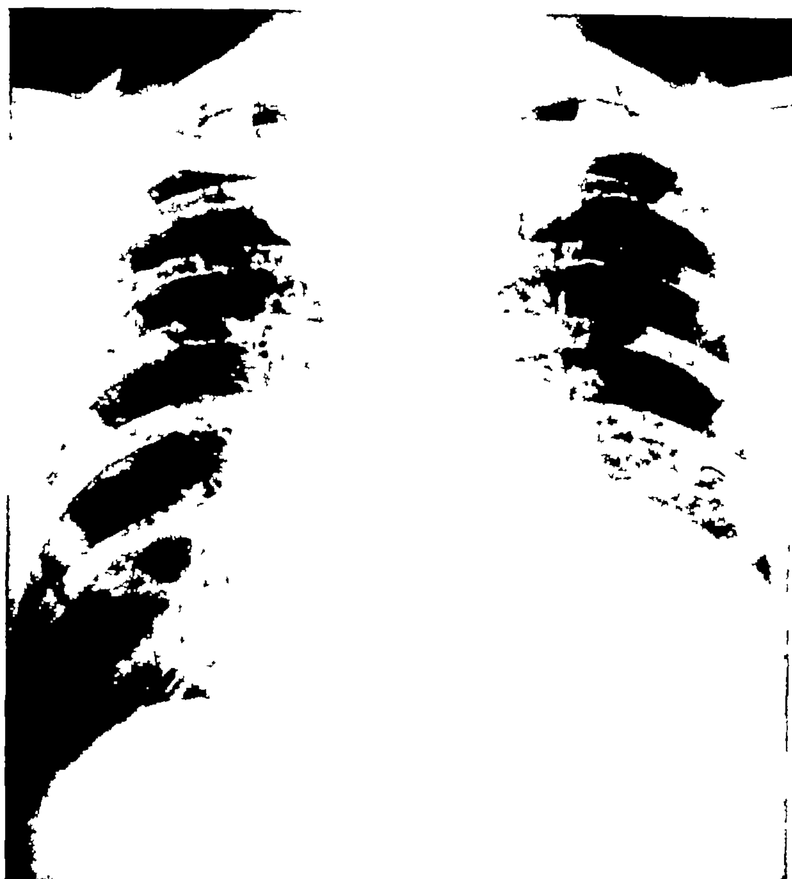


FIG 319 Notching of the ribs, absence of aortic knob and left ventricular enlargement in coarctation of the aorta

splanchnicectomy we have picked up a slight delay in the rise of the pulse wave in the groin There are undoubtedly some mild forms of coarctation in which femoral pulses are strong and in which there is no or very slight rise of systolic pressure above 140 mm of mercury These patients probably need no treatment Since the occlusion is usually situated at the point where the ductus arteriosus joins the aorta and is distal to the left subclavian artery, hypertension occurs only above this level Collateral circulation is evident in the scapular and cervical branches of the subclavian artery, in the lateral and dorsal branches of the abdominal aorta and through the upper two intercostals to the lower intercostals (fig 318) These enlarged collaterals can sometimes be best seen and felt between and below the shoulder blades When the stenosis is proximal to the left subclavian artery, there is a difference in the pulse and blood pressure between the right and left arm (see p 465)

Radiologically, the scalloping or notching of the lower ends of the ribs, produced by the enlarged and tortuous intercostal arteries, can be detected (fig 319) In the oblique views, the aortic arch cannot be traced Other anomalies, such as bicuspid valvulitis with aortic insufficiency and patent ductus arteriosus, may be associated with coarctation

If the exact location and length of the stenotic segment need to be visualized to better prepare for a transplant, visualization of the aorta can be car-

ried out through the carotid and brachial arteries or through the transverse aorta

Pheochromocytoma

Only about one third to one fourth of the patients harboring a pheo chromocytoma show paroxysmal attacks of hypertension on the other hand, many essential hypertensives exhibit attacks of anxiety with paroxysmal rises in blood pressure as do some patients with attacks of hypoglycemia which stimulates medullary adrenal activity

A typical attack consists of sudden pallor excessive sweating, dyspnea chest and abdominal pain throbbing headache nausea, vertigo weakness and tremor During this attack the blood pressure which may have been known to be normal rises to pressures of 200 mm Hg systolic and 110 mm Hg diastolic, or higher Blood sugar and basal metabolic rate may rise Table XII summarizes the principal symptoms in 18 cases of paroxysmal hypertension

Table XII

FREQUENCY OF OCCURRENCE OF THE PRINCIPAL SYMPTOMS IN 18 CASES DURING PAROXYSMAL HYPERTENSION*

SYMPTOMS	NUMBER OF CASES
Blanched or mottled cold extremities	17
Palpitation	17
Nausea	16
Sweating	14
Vomiting	13
Headache	10
Pulmonary edema	9
Precordial pain	8
Distention of neck veins	5
Dilated pupils	4
Body tremors	3
Dizziness	2

* Howard, J E. and Barker W H Paroxysmal Hypertension and Other Clinical Manifestations Associated with Benign Chromaffin Cell Tumors (Pheochromocytoma) Bull. Johns Hopkins Hosp. *61* 371 1937

Since the symptoms are essentially due to the release of mixtures of norepinephrine and epinephrine followed by a state of anxiety a reproduc tion of these attacks under the direst observation of the examiner may be of great diagnostic value This is best done by the histamine test of Roth and Kvale (0 05 mg I V) but only if the basal blood pressures are below 170/110 mm Hg. It is important that prior to the test no sedation be given for at least 48 hours and no hypotensive drugs be given for at least two days otherwise false positive or false negative results will be obtained [55] Long acting hypotensive drugs may have to be discontinued for weeks

Since patients may die in a spontaneous or induced attack, Regitine or benzodioxane should always be available In a 55 year old Russian tailor, who had attacks of angina, excessive sweating, cold, clammy extremities, a coarse tremor with bradycardia and paroxysmal hypertension, exploration was refused by an internist, chiefly because of the absence of tachycardia At that time the effect of norephinephrine, which does cause bradycardia, was unknown When discharged to his home, the patient died in an acute attack with a cerebral hemorrhage Autopsy revealed a walnut-sized pheochromocytoma, which could have been readily removed

Since the majority of such patients exhibit a continuous hypertension with slight or absent paroxysmal rises of pressure, adrenolytic drugs, preferably Regitine in 5 mg doses given intravenously, are employed to see how much of a fall in blood pressure is obtained False positive tests are not infrequent, especially in uremic patients, and if the drug is given intravenously more false positive hypotension is encountered As Grimson pointed out,[24] Regitine shows fewer side effects than piperoxane (benzodioxane), and the reduction of blood pressure lasts longer Curiously enough, piperoxane also produces an antidiuretic effect, but only in cases of pheochromocytoma, and thus can be used as confirmatory evidence of a suspected chromaffin tumor.[56]

Of course, the direct evidence of increased catecholamine excretion in the urine is most conclusive, the colorimetric test of Sulkovitch also offers much promise [57]

The greatest diagnostic difficulties occur when patients with persistent hypertension harbor an unsuspected pheochromocytoma, and the table of Smithwick and his co-workers offers useful hints for suspecting such a tumor (Table XIII)

Table XIII

SYMPTOMS, SIGNS AND FINDINGS SUGGESTIVE OF PHEOCHROMOCYTOMA IN PATIENTS WITH PERSISTENT HYPERTENSION*

SYMPTOMS AND SIGNS	INCIDENCE IN 11 PHEOCHROMOCYTOMAS OBSERVED (PER CENT)	INCIDENCE IN 107 PHEOCHROMOCYTOMAS REPORTED (PER CENT)	INCIDENCE IN 100 CASES OF ESSENTIAL HYPERTENSION (PER CENT)
Excessive sweating	90	52	2
Vasomotor phenomena	90	47	0
Elevated temperature	78	70	10
Normal cold pressor response	73	63	22
Fasting blood sugar <120 mg	64	61	13
B M R +20 per cent	60	57	5
Postural tachycardia	55	—	15
Postural hypotension	44	50	3
Glycosuria	36	50	4
Paroxysmal attacks	36	75	0

* Smithwick, R H , Greer, W E R , Robertson, C W and Wilkins, R W Pheochromocytoma New England J Med , *242* 252, 1950

Pheochromocytoma may produce a malignant phase of hypertension and in such cases one would naturally suspect secondary renal arteriolar sclerosis This is however not always true Palmer and Castleman s and Pickering s cases (cited in 2c) exhibited no vascular lesions in the kidney even though the hypertension may have persisted after removal of the tumor In the careful analysis of W S Peart,[2a] the cause of the residual hypertension, which occurs in some cases after the removal of a pheochromocytoma, is uncertain Both renal and extrarenal factors may be present I have stressed this interesting phenomenon in discussing the "vicious circle in hyper tension (p 476) a concept which is of considerable significance for the surgeon [1]

Unilateral Renal Disease

The obvious surgical importance of detecting, localizing and removing a nonfunctioning kidney which is the cause of hypertension needs to be stressed since many urologists and internists have voiced scepticism that hypertension can thus be eliminated In our own experience the congenital hypoplastic kidney the unilateral atrophic pyelonephritic kidney inter mittent obstruction of the renal artery due to nephroptosis radiation neph ritis following deep roentgen ray therapy an embolic infarct and atheroma tous stenosis of the renal artery can all cause hypertension we have also reported two traumatic injuries to the kidney causing hypertension [46] In addi tion to a careful history intravenous pyelogram and aortograms may help to localize the involved kidney Again as pointed out by Pickering,[2c] the re moval of the original cause may abolish the hypertension in roughly one half of patients in whom nephrectomy is done for unilateral pyelonephritis But just as after removing a coarctation of the aorta or a pheochromocytoma, mecha nisms may come into play which maintain hypertension even when the origi nal cause is removed Particularly the work of Goldring and Chasis [58] who maintained that nephrectomy was of no value for unilateral renal disease with hypertension and who did not believe that pyelonephritis caused hyper tension at all has deterred many of our internists and urologists from look ing at hypertension as a removable, surgical disease Our experience indicates that the height and duration of hypertension and the presence of the ac celerated malignant phase determines whether or not nephrectomy or nephrectomy and bilateral dorsolumbar sympathectomy is going to help these patients I have seen a number of pediatric residents fiddle with juvenile hypertensions in 4 to 8 year old children until the malignant phase has made their cases hopeless and inoperable Brust and Ferris have indicated that the reversibility of the hypertensive process may be predicted by the use of a ganglionic blocking agent [59]

Generally speaking, juvenile hypertension speaks for congenital hypo-plasia of the kidney atrophic pyelonephritis or coarctation of the aorta An occasional pheochromocytoma may occur The absence of positive urine

cultures, which need much skill and care, or a marked deformity of the renal pelvis does not rule out pyelonephritis, pyelonephritis does not seem to cause hypertension until vascular obstruction or incomplete infarcts are produced, secondary to the inflammatory reaction [13] The same is true of periarteritis nodosa

Bilateral Renal Disease

Nephritis, types I and II, post-toxemic hypertension, chronic pyelonephritis, periarteritis nodosa and polycystic kidneys all seem to be typically renal types of hypertension, in which the surgeon is not supposed to have any interest As a matter of fact, in 1947 Fowler and I[46] presented case histories to indicate that a group of patients with organic renal damage showed good response to splanchnicectomy, whereas a so-called "neurogenic" group failed to respond because, as we suggested then, of the presence of hypothalamic-pituitary stimulation Particularly, the post-toxemic hypertension in which there is glomerulonephrosis,[60] the pyelonephritic hypertension, the streptococcus nephritis followed by hypertension, the "rheumatic" hypertension and a case of periarteritis nodosa with hypertension were described and shown to respond to surgery [46 50]

What is the common denominator in these cases? In the first place, the primarily renal hypertension may later develop an increased vascular reactivity [61] Then also a vascular obstruction of the interlobar, interlobular and arcuate arteries, resulting in incomplete cortical infarction, has been described in detail in such renal inflammations which are accompanied by hypertension [13] Third, when these renal infections enter the malignant phase, they become insulin resistant and Regitine sensitive, indicating adrenal participation Thus a pure "renal" hypertension in the chronic phase really does not exist

TREATMENT

The objectives of treatment are obvious (1) A removable cause of hypertension is looked for, such as coarctation, pheochromocytoma, Cushing's syndrome or unilateral renal disease, and attacked surgically before a "vicious circle" prevents a reduction of blood pressure, even though the original cause has been removed It should be emphasized that even removal of urinary obstruction by prostatectomy may reduce hypertension (2) In case of essential hypertension, reduction of the diastolic pressure or the arrest of a rising diastolic pressure slows down progressive vascular damage and may avert the malignant phase (3) In the early malignant phase with fairly adequate renal function, reduction of blood pressure may bring on regression to a premalignant phase

In a surgical monograph, the dietary and drug therapy needs only a brief enumeration of principles The surgeon, however, must be aware of these methods, and a combination of medical and surgical measures may give an optimal chance for the patient

Dietary Measures

The low salt diet is a time honored method in the treatment of edema and nephritis Frederick Allen used severe salt restriction in 180 advanced cases of hypertension and reported failure in 55 per cent of them [62] Kempner's rice diet,[63] which consists of 20 Gm of protein 5 Gm of fat 200 mg of chloride and 150 mg of sodium—a total of 2 000 calories—reawakened interest in this field The psychic effect of a distant Mecca the sanatorium treatment, the strict enforcement of a low salt intake all contribute to its value and the author has seen patients—usually women—adhere fanatically to a monotonous, unappetizing regime which may precipitate uremia Improvement may be expected in one fourth to one half of the patients treated, but, except in very rigid or obsessive patients adherence to the diet for prolonged periods is difficult

There appears to be no advantage of the rice diet over the 200 mg low sodium diet, and as long as the sodium content is not raised above 500 mg per day the hypotensive effects are maintained even if fat and protein are added [64] Obviously in edematous patients with heart failure or in malignant hypertension this is a worth while measure if a careful eye is kept on the blood urea

The surgeon should be cognizant of the low sodium syndrome of weakness nausea, vomiting and hypotension which may appear following surgical procedures for hypertension especially in patients who had been on a low sodium diet before operation The patient with an unresponsive adrenocortex or with inadequate adrenocortical reserve readily enters a phase of negative nitrogen balance [65]

In a study of the cortico-adrenal factor in hypertension I suggested several years ago that since only a certain percentage of patients respond to salt restriction, these would be the ones in whom cortico-adrenal hyperactivity is present, and they might be recognized by their resistance to insulin [15] I am not aware that such a correlation has ever been made but it is apparent from all reports that some hypertensive patients respond better than others to sodium restriction and, in fact, the hypertensive patient can stand sodium withdrawal much better than can the normotensive person Certainly blood sodium-potassium ratios should be obtained to detect aldosteronism

Recently the changing patterns of sodium metabolism in hypertension were intensively studied by D M Green [66] In the early stage there is increased urinary output of sodium and water under load an increased appetite for salt and a progressive rise in blood pressure In the late stage there is normal or decreased sodium and water output under load, normal or subnormal appetite for salt and a sustained level of hypertension Success of any treatment will depend on attacking the first phase and a standardized salt loading test is said to differentiate between the two phases This consists of administering 5 per cent sodium chloride, 100 cc per square meter intravenously for 20 minutes These two phases may relate to reversible and ir

reversible changes in hypertensive cardiovascular disease and seem to be present in all types of hypertension

Salt restriction then may aid the patient, but in only certain phases and types of the disease, and it should be administered with great caution in the presence of renal damage Its wholesale use is senseless and may be dangerous, especially before operations

Drug Therapy

The present enthusiasm for hypotensive therapy has so permeated the management of hypertension by the general practitioner and by the internist, that vascular surgical services, which carried in the past a heavy schedule of operations for hypertension, now only see an occasional and probably irreversible case referred for operation It behooves us then to examine this pharmacologic approach, its value and its limitations

The drugs may be central sympathetic depressants at hypothalamic level, such as Rauwolfia preparations, they may act on the ganglionic synapse like methonium, pentolinium or mecamylamine, they may influence the adrenergic end organs like Dibenzyline, or, they may stimulate the Bezold-Jarisch reflex through the carotid sinus nerves and the vagus, like the veratrum preparations Hydrazaline acts on the central nervous system, binds vasopressor substances and perhaps toxic metals by chelation and produces renal vasodilatation.

The combination of drugs has assumed mammoth proportions The side effects, such as postural hypotension, a lupus erythematosus type of disease, pulmonary fibrosis and drug tolerance, call for a skillful application of these drugs which are unquestionably potent and toxic but effective The greatest objection to their prolonged use lies in the fact that the patient who responds to this treatment is the same one who would obtain maximum benefit from surgery By prolonged drug therapy the irreversible component of his disease is increased by the time he is deemed suitable and ready for surgery There is, of course, a real place for these drugs both before and after the operation, and it is sufficiently clear that they are far more effective after the operation

Psychotherapy

In our hospital study, a psychiatric social service worker interviews every patient, and remarkable case histories have accumulated from these interviews.* If the patient is approached tactfully, he gladly unburdens himself, and this itself relieves some tension For those in whom a severe psychoneurosis or psychosis has been discovered, rapid psychotherapy may be carried out preoperatively and postoperatively, such as that outlined by Alexander and French [54] But we have never seen any sustained hypotensive

* See a sample in the appendix, page 532

effect of psychotherapy alone In the studies by Wolf and his associates,[2f] of 114 hypertensive subjects 14 lost all evidences of hypertension when treated by repeated interviews in the hypertension clinic The purposes of the interviews were (1) to enable the patient to recognize that he felt threatened and hence angry and anxious (2) to indicate how when he did feel threat ened he might deal with the danger by more direct and appropriate action rather than by repression and (3) to help him feel more secure " The case reports of these patients reveal elevations and depressions of blood pressure during the interviews, but one fails to get the impression that their cardio-vascular renal disease has been influenced In our own experience patients referred to the Institute for Juvenile Research of the University of Illinois for psychotherapy emerged from the Institute after six months to a year much happier but not less hypertensive In fact, just as during drug therapy a year was lost

Surgical Treatment

Medical opinion, which of course is necessary for a successful team work in this field varies a great deal regarding the indications for surgical treatment Obviously everyone agrees that when a removable cause is found such as coarctation of the aorta unilateral renal disease, pheochromocy toma, a cortico-adrenal adenoma or hyperplasia the surgeon should proceed without delay Attention should be called again, however to the fact that hypertension caused by these factors may persist if it has reached the self perpetuating second phase or the accelerated malignant one In such cases splanchnicectomy should be added to a nephrectomy or to a removal of a pheochromocytoma, even in a Cushing s syndrome The question naturally arises as to the possibility of a preoperative recognition of such a state

In the case of unilateral renal disease biopsy of the opposite kidney may indicate that the blood pressure will not return to normal We have published such cases previously In the case of hypoplastic kidneys of young children the malignant phase will progress to uremia and death in spite of an added splanchnicectomy In pheochromocytomas, Peart[2a] has made an attempt to find the cause of sustained hypertension after removal of the tumor he could not find renal damage or any other definite cause in all cases although pyelonephritis was suggested as a factor

In addition to a secondary renal factor the pressure regulating mecha nisms in prolonged continuous hypertension may need more investigation The barostat, which must be in or around the vasomotor center is under continuous bombardment during hypertension and must be set at a higher level in order for the buffer nerves not to get exhausted All studies relating to the carotid sinus mechanism in hypertension have been negative so far in fact, the electrical impulses, the rate of firing, are greatly increased trying to inhibit the vasomotor center [67] If then the barostat has been set higher as a result of continuous hypertension, removal of the original cause such as a pyelonephritic kidney may still leave this "neurogenic" hyper

tension intact, which is essentially due to an adaptation and decreased sensitivity of the baroceptors Such baroceptor fatigue is denied by Kezdi and Hilker[8] who believe that first functional and later organic rigidity of the vessel wall leads to decreased inhibition of the vasomotor center Whether the vasomotor center may become more reactive and more sensitive as a result of cerebral circulatory involvement is uncertain, but is a distinct possibility.

Sympathectomy

In "essential hypertension" the majority of patients deemed suitable for surgery have undergone sympathectomy In our clinic the indications for sympathectomy, which my co-workers and I established in the early 1930's and to which we still adhere, are as follows

(1) In group 1, the patients are below 40 There is minimal or no detectable organic damage except retinal vascular sclerosis The casual diastolic blood pressures are above 100 mm of mercury but the blood pressure falls to normal on complete bedrest or barbiturates Tetraethylammonium chloride reduces diastolic blood pressure below 90 mm of mercury *Such patients should be placed on small doses of barbiturates or Rauwolfia, but if the retinal vascular changes progress, immediate dorsolumbar sympathectomy is performed* This indication is based on our experience that hypertension developing at such an age is progressive, especially if both parents have had hypertension, that the results of a limited sympathectomy, which often can be performed in one stage, are excellent, and that simple psychotherapy by the patient's own physician or by a psychoanalyst, a change of occupation, avoidance of life stress may result in a happier individual but in no arrest in the progress of the disease.

In the late 1930's and early 1940's, a small group of such patients were operated on Since the patients are asymptomatic and since today drug therapy, especially Rauwolfia preparations, is in great vogue, hardly ever does one now encounter such a group, either in institutional or in private practice. Nevertheless, this group has done exceedingly well in 5 to 10 year follow-up studies Of 21 patients followed for 10 years or more, 16 showed a worth while result. Certainly evidence of increasing arteriolar damage in the retinal and renal areas has been lacking

(2) To group 2 belong patients with arteriolar damage in all organs with a demonstrable vasomotor component, as demonstrated by a tetraethylammonium chloride response of the diastolic level Of course, one would like to see this "diastolic floor" drop to 90 or 100 mm of mercury, but a drop from 140 to below 120 mm is good evidence of a sufficient vasomotor component superimposed on organic damage There are, of course, thousands of middle-aged arteriosclerotic hypertensive persons who do well on reassurance, rest periods, moderate salt restriction, phenobarbital or minimal doses of Rauwolfia Should their diastolic pressure rise, however, I have advised a sympathectomy of the lumbodorsal type, under skillful medical manage-

ment with the object of halting the arteriolar disease and preventing the malignant phase Of 63 patients who have had 10 to 20 year follow ups 42 have had worth while results in our hands

(3) In group 3 are patients with a high fixed diastolic pressure which can not be lowered below 120 mm of mercury by any depressor drug There are large recurrent hemorrhages and papilledema There may be congestive or anginal heart failure and cerebrovascular accidents have occurred Renal function is impaired but there is still at least 15 per cent of P S P * excretion in 15 minutes and a concentrating ability of the kidney to 1 015 specific gravity This is a premalignant or early malignant phase of hypertension which is still acceptable for operation and where the limiting factor to sur gery is renal function Since the definition of the accelerated phase is so important, George Perera should be quoted verbatim We should suspect an accelerated phase when our patient with primary hypertension develops signs of progressive renal involvement, accompanied frequently but not always by retinopathy and papilledema Our clinical diagnosis will be about 90 per cent accurate and only necropsy will improve our batting average [68] He as we has seen patients with grades 2, 3 or 4 retinopathy go into renal failure and, conversely into papilledema and retinal hemorrhages a grade 4 retinopathy is not infrequently seen with comparatively adequate renal function

The question whether the accelerated phase represents some super imposed pathogenetic factor (Perera)[68] or whether it is simply the result of continuously rising, severely elevated diastolic pressure (Pickering)[2c] is im portant here since a prompt surgical reduction of diastolic pressure *reverses* this accelerated phase into a severe benign phase this indicates that the pressure itself is responsible for the necrotizing arteriolitis, exudates and hemorrhages The experimental studies of Byrom[69] indicated severe arterial spasm provoked by the gross rise of intravascular pressure as the cause of fibrinoid necrosis of the wall, hemorrhages and exudates reducing the pres sure promptly abolished the vasospasm

This nonneurogenic vasospasm is said to be due to a stretch reflex and not influenced by sympathectomy unless it reduces blood pressure All observers have seen the rapid regression of papilledema and the ceasing of hemorrhages after a sympathectomy in the malignant phase In our experi ence however the splanchnicectomy has to be combined with sympathec tomy from the second dorsal to the third lumbar ganglia The indication for sympathectomy here is acknowledged by Pickering[2c] who feels that an effec tive lowering of blood pressure by all available means is imperative

The determining factors of the appearance of the malignant phase are the height of the diastolic pressure and also the rate at which it rises Obvi ously local factors producing vulnerability of the vessel wall are also im portant

Thus if diastolic pressure rises especially in males whose prognosis is obviously worse no time should be wasted before an extensive sympathec

* Phenolsulfonphthalein

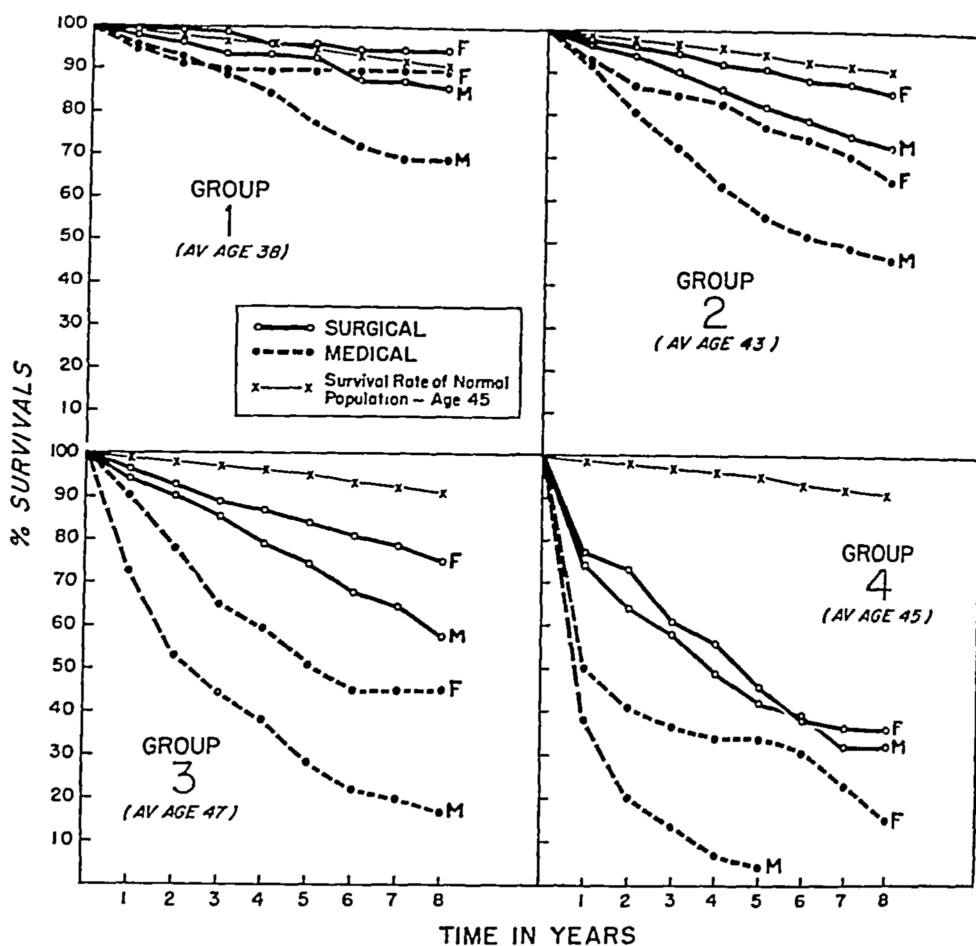


FIG 320 Survival curves for 2,227 hypertensive patients separated into four groups, comparing male and female survival rates (Smithwick, et al Hypertension and Associated Cardiovascular Disease J A M A, *160* 1023, 1956)

tomy is undertaken Of 16 patients, 7 were alive after 5 years, certainly all were salvaged from certain death

The five year mortality rates of 1,118 male and 1,109 female patients classified into four groups (groups 3 and 4 are our group 3) have been obtained by Smithwick and his co-workers [70] The mortality rates were significantly better in all four groups of surgically treated male patients, and in groups 2 and 3 of the surgically treated female patients (fig 320) The four groups of Smithwick are not quite the same as those of Keith, Wagener and Barker, but essentially they are the same as our three groups, our third group containing groups 3 and 4 of Smithwick There are many other numerical values attached by different authors to various grades and phases of hypertension.

One can operate on essential hypertension too early, adolescent hypertension and labile intermittent hypertension in young women or even in young men with no vascular damage require no surgery Middle-aged arteriosclerotic patients with a stable diastolic pressure, who observe weight control, moderate exercise and perhaps a mild cortical or hypothalamic sedation, are not subjected to surgery Parenthetically, much unnecessary surgery on this group was done in some clinics, thus creating a deserved reaction against unjustifiable operations One is often pressed to operate on patients too late

in the full blown malignant phase in which renal failure is imminent and can be precipitated by operation by salt restriction or by hypotensive drugs In tracing the development of the individual case to this terminal phase, one is deeply impressed by the opportunity lost to operate on these patients when they still have some renal reserve

Method of Evaluating Results Smithwick and his co-workers have used survival rates in four groups of male and female hypertensive patients [70] Our criteria for *worth while* and *not worth while* results have been arrived at by examining four factors in a recently completed follow up [1] a 20 mm of mercury reduction of diastolic blood pressure a definite improvement in cardiac size and in the electrocardiogram, partial or complete regain of earning capacity and subjective relief from symptoms Any patient who shows improvement in at least three out of these four catagories has been classified as having received benefit In the first group of hypertensives we require normal or almost normal blood pressures and full working ability In the second group we have to see stabilization of diastolic pressures, regression of vascular damage full working ability and symptomatic relief In the premalignant to malignant phase we are satisfied with arrest of the disease regression of retinal findings restricted working ability and symptomatic relief

In the case of Wm. T., a grade 3 hypertensive man the heart size diminished and stayed so for three years after splanchnicectomy (fig. 321) Charles F., a 48 year old hypertensive diabetic patient, was decompensated without digitalis He lived three years without digitalis and died of a ruptured aneurysm of the basilar artery (fig. 322) Electrocardiograms improve very fast

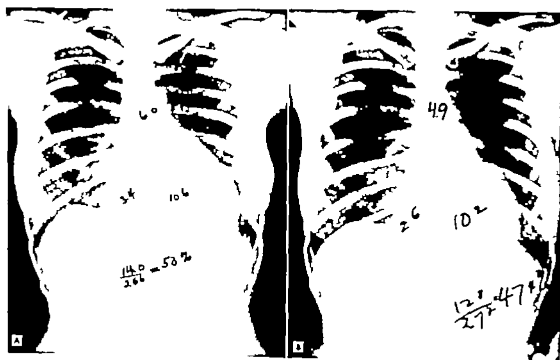


Fig 321 Two meter chest films before (A) and three years after (B) bilateral splanchnicectomy (Wm. T., grade 3 hypertension.)

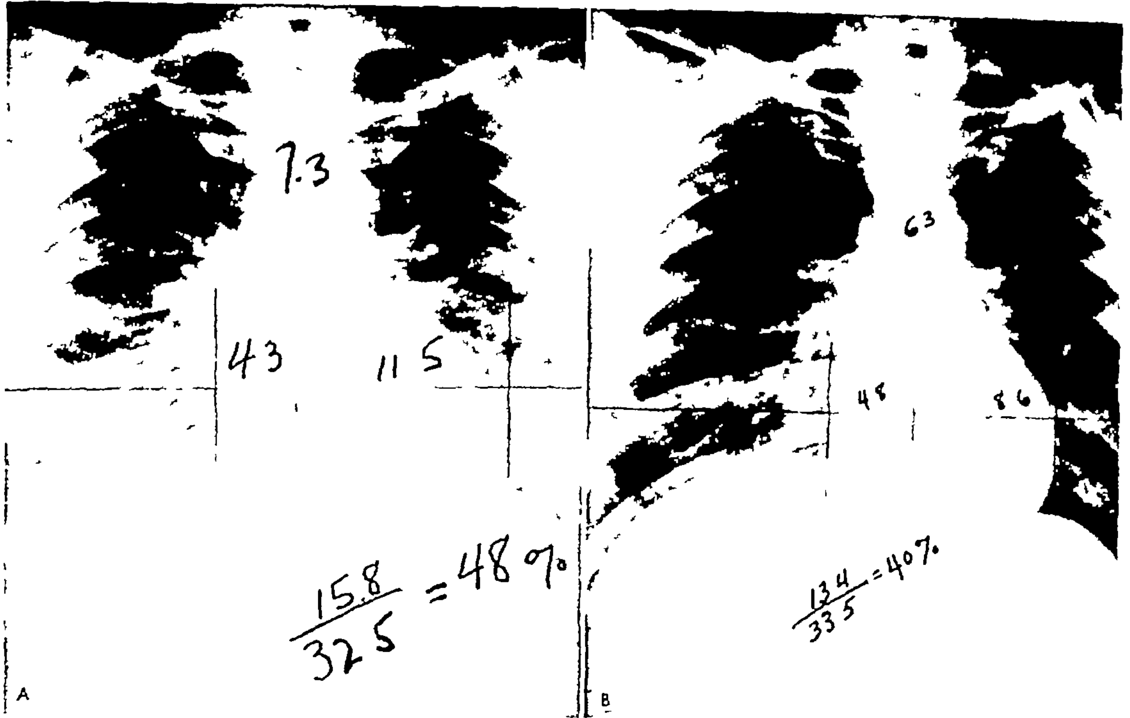


FIG 322 Two meter chest films before (A) and one year after (B) splanchnicectomy in Ch F, a diabetic hypertensive patient in decompensation He died of a subarachnoid hemorrhage three years after surgery

I

II

III

IVF

FIG 323 Electrocardiogram before (A) and eight months after (B) splanchnicectomy in a 44 year old hypertensive man, Leonard R He died five years later of an acute coronary occlusion after violent exercise

after splanchnicectomy as shown in figure 323 but this did not prevent this man from dying five years later of a coronary occlusion after violent physical exercise All three patients have had definite remission of their disease but in the 10 year follow up they are classified as failures having died of vascular accidents

In our recent study with McDonald and Harridge [1] a total of 105 patients were traced, who were operated on for diastolic hypertension between 1934 and 1946 Of the 105 patients followed from 10 to 22 years 20 died, 61 were better and 24 were worse (fig. 324) In order to determine their vascular status and compare it with the preoperative one a severity index was established according to the criteria shown in Table XIV In each of the five panels *i e* diastolic pressure cardiac function renal function cerebral involvement and eyegrounds, four grades can be registered Thus in a terminal phase of malignant hypertension with early vascular damage in eyegrounds brain heart and kidney a severity index from 3 to 8 is found When the preoperative indices of 79 patients were compared with the postoperative indices obtained by personal examination or questionnaire (fig. 325) it is apparent that the severity indices have become lower in the total group indicating an over all reduction in severity Since such a frequency polygon does not reveal improvement of the individual but of the whole group we have compared the severity indices of the individual and have thus reached the figures shown in figure 324 In regard to individual symptoms and signs, Table XV indicates that over a period of 10 to 22 years roughly 60 per cent of these hypertensive patients have maintained an improvement Since all of these patients were operated on before the advent of the newer hypotensive drugs comparatively few have received them and in fact most of them are on no medication at all

Side Effects and Complications Fowler and I[71] studied the untoward sequelae of splanchnicectomy for hypertension and summarized our findings Of the *cardiac complications* acute coronary insufficiency and coro-

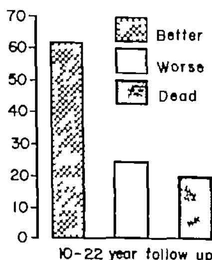


FIG 324 End results of surgical treatment of hypertension after 10 to 22 years, based on a comparison of the severity indices of the individual before and after operation (de Takats, McDonald and Harridge The Vicious Circle in Hypertension. Surgery *43* 113 1958)

Table XIV

SEVERITY INDEX OF DIASTOLIC HYPERTENSION*

	GRADES			
PANELS	1	2	3	4
Diastolic pressure (in mm Hg)	95–110	111–125	126–140	<140
Cardiac function	Early myocardial change, slight enlargement	Marked myocardial damage, heart over 50 per cent cardiothoracic ratio	Angina or controlled compensation	Coronary occlusion, congestive heart failure
Renal function (15′ P S P in per cent)	35–25	24–20	19–15	14–10
Cerebral involvement	0	Severe hypertensive headache	Old stroke	Fresh stroke or encephalopathy
Eyegrounds	Narrowing or spasm	A V nicking and much sclerosis	Hemorrhage and exudates	Papilledema

* de Takats, G , McDonald, G O and Harridge, W H Surgery, *43* 113, 1958

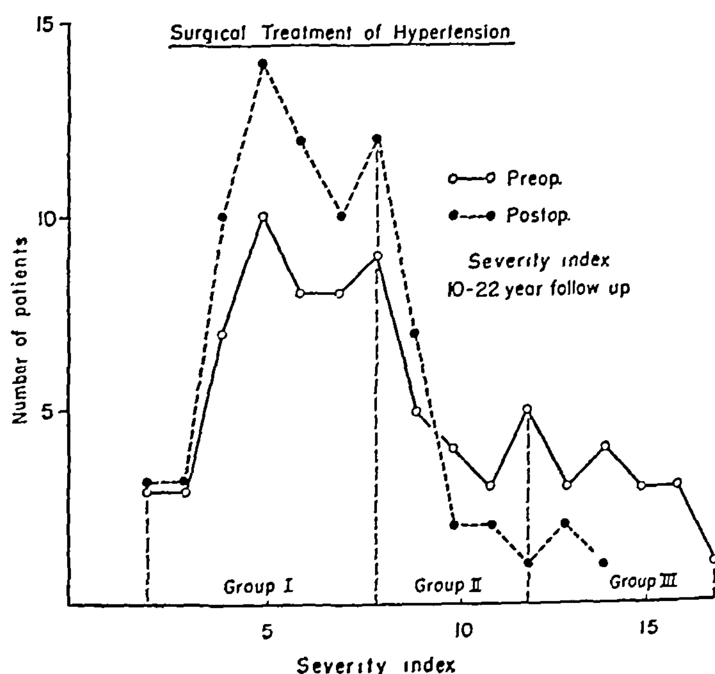


FIG 325 Frequency polygon of severity indices before and 10 to 22 years after dorsolumbar sympathectomy. Note that group 1, with the index of 3 to 8, has increased after the operation, whereas in groups 2 and 3 there are fewer cases left postoperatively The severity of the total group has decreased (de Takats, McDonald and Harridge The Vicious Circle in Hypertension Surgery, *43* 113, 1958)

nary thrombosis occurred, with one fatality. An alert resident staff aided by competent internists can readily tide patients over an acute heart failure, usually brought on by prolonged hypotension, by poor anesthesia leading to atelectasis and by accumulation of carbon dioxide Slow or incomplete re-

placement of lost blood may also be responsible Overhydration of a patient who normally retains sodium and water as a response to a major surgical stress is to be avoided Electrocardiographic evidence of an old myocardial infarct is no contraindication to surgery but both during and after the operation, hypotension below 110 mm of mercury should be actively treated the best method in our opinion is with norepinephrine A recent myocardial infarct is obviously a definite contraindication to a lowering of blood pressure We have preferred to wait six months following an acute myocardial infarct, but naturally some of these infarcts, possibly 10 per cent are silent and the date of their occurrence is unknown

Table XV

IMPROVEMENT FOLLOWING SURGICAL TREATMENT FOR HYPERTENSION

SYMPTOM OR SIGN	IMPROVEMENT IN PER CENT
Headache	75
Palpitation, Dyspnea, Angina, Ankle edema	60
Urinary frequency	—
Electrocardiogram	51
Heart size	47

* de Takats, G McDonald, G O and Harridge, W H The Vicious Circle in Hypertension. *Surgery 43 113 1958*

Of *cerebral complications* we have encountered 6 in 100 patients undergoing 200 operations These represent a cerebral vascular insufficiency in arteriosclerotic brains Most of them exhibited a recurrence of a previous neurologic lesion such as an abducens paralysis or a hemiplegia but fortunately *in none of them did the neurologic lesion persist* As an emergency procedure, an immediate procaine block of the cervical sympathetic trunk,[72] first on the affected side and six hours later on the opposite side has been of definite benefit

There has not been a single case of uremia or acute *renal failure* not only in this particular group but in our total series of over 600 patients this is because of the rejection of patients for sympathectomy whose renal impairment is marked Postoperative deaths from uremia, however have been reported by others Since splanchnicectomy in our experience is futile after renal participation in hypertension has become marked there seems to be no reason to subject such patients to an acute hypotension

Pulmonary complications are of course common and are much more frequent in the transthoracic than in the retropleural type of approach Good anesthesia is of course, very important but is not always available when too many residents are supervised by a single trained anesthesiologist. Induction with nitrous oxide followed by ether is a very satisfactory type of anesthesia,

and tracheal intubation is always necessary When the pleura has been entered either purposely or inadvertently it is safer to leave mushroom catheters in the pleura for 48 hours to utilize continuous suction

Intercostal neuralgia may be a highly disturbing complication, and occurs both after splanchnicectomy and after thoracotomies Rib spreaders seem responsible for some of the traction neuritis, but the cut proximal end of the rib, postoperative scarring, hemorrhage or ischemia of the posterior root ganglion and pressure of the retractors on the somatic nerves may all be responsible In this small, particularly studied series of 100 patients, intercostal neuralgia occurred in 16 Neither section nor crushing of the intercostal nerves is helpful, nor have the massive (1000 γ) doses of vitamin B_{12} been beneficial The complaint subsides within six weeks, but in an occasional patient the intercostal nerve or the genitofemoral nerve may have to be freed up from scarring Paravertebral block to the proximal end of the divided sympathetic trunk occasionally helps, just as in the postsympathectomy neuralgia after lumbar sympathectomy

Myalgia, a weak back and osteoarthritis of the spine are occasionally troublesome, and the position of the patient on the table may be responsible

Injury to the thoracic duct has not occurred in any of our cases, but we are continuously watching for it Ligature of the duct is the wisest course when chyle is seen to exude from the wound

Postural hypotension and postural dyspnea are temporary side effects when the lumbar chain is resected together with splanchnicectomy Hyperhidrosis in nondenervated areas is only troublesome in total or subtotal sympathectomies For further details, our study with Fowler may be consulted [71]

Adrenalectomy

The use of total or subtotal adrenalectomy for Cushing's syndrome is clearly established Sprague, Kvale and Priestley[73] reviewed their experience with 50 cases of Cushing's syndrome, in which no functioning cortico-adrenal tumor was found but in which bilateral adrenal hyperplasia was present In the last 27 cases there has been only one postoperative death, and much of their success can be readily attributed to the skillful use of cortisone and desoxycorticosterone acetate Of the 41 patients living in 1953, 40 were in a state of satisfactory remission, but it is emphasized that recurrences may be expected after the passage of more time When a cortico-adrenal adenoma (fig 307) is present, however, the contralateral gland is usually atrophic. When the first adrenal gland which is explored is large but contains no adenoma, the patient has adrenal hyperplasia and the other gland will be large also Most importantly, however, if the first gland explored is normal in size, this adrenal and its opposite are not hyperplastic and there is no contralateral tumor [74] Subtotal adrenalectomy is still the method of choice here if there is a full-blown Cushing's syndrome or if the so-called essential hypertension shows marked increase in cortico-adrenal activity The recent recog-

nition of primary aldosteronism, which is cured by adrenalectomy is worthy of emphasis The adenoma was not palpable in one case and only the severe hypokalemia and metabolic alkalosis suggested the diagnosis [75]

This brings us to the recent procedure of two groups, one at the Peter Bent Brigham[76] and the other at the University of Pennsylvania [77] who advocate subtotal to total adrenalectomy for severe essential hypertension In analyzing the results of these two groups it seems that the measurable benefit is overwhelmingly on the basis of sodium and water diuresis in the Brigham series the best results have been obtained in patients with dyspnea congestive heart failure and intracerebral edema The Pennsylvania group has combined adrenalectomy with splanchnicectomy and feels that this com bination has improved the end results Unfortunately however they do not advise adrenalectomy when less than 20 per cent of the injected phenol sulfonphthalein is excreted in 15 minutes this rule excludes exactly the type of patient for whom one would like to extend the limit of operability

While splanchnicectomy in our hands has a mortality of less than 1 per cent, 24 per cent died after adrenalectomy a mortality far too high in a type of operation in which results are unpredictable It is true that substitution therapy has made great strides and that daily maintenance with cortisone desoxycorticosterone acetate and salt is possible for many patients who have been operated on for Cushing s syndrome or for certain forms of cancer In essential hypertension however we can only see such an indication where the cortico-adrenal factor as evaluated by our simple clinical tests is demon strably predominant.

A recent analysis[78] of the results obtained by 90 to 100 per cent adrenal ectomy combined with a subdiaphragmatic sympathectomy and splanchni cectomy of the Adson type indicates that the mortality now has been reduced to 5 per cent because of eliminating the advanced renal cases and that re-placement therapy is becoming more and more standardized The authors[78] advocate this procedure as having less morbidity than the more extensive sympathectomy Their data show no convincing evidence that adrenalec tomy should be added to sympathectomy in the essential hypertensive patient

Hypophysectomy

It has been known for some time that experimental renal hypertension can be abolished by removal of the anterior lobe (but not the posterior) of the pituitary [79] Recently the Swedish surgeon Olivecrona with Luft re ported cases of severe diabetic retinopathy subjected to hypophysectomy they later added some cases of hypertension to their series [80] This is truly a drastic procedure although substitution therapy following removal of the pituitary gland is said to be simpler than that after adrenalectomy At the present writing, I would hesitate to recommend such a procedure in the cases of malignant hypertension for which it was advocated

Unilateral Nephrectomy

There is an increasing awareness on the part of the internists of the role that chronic pyelonephritis plays in hypertension As has been stated before, pyelonephritis does not lead to hypertension in the acute phase, and does so in the chronic phase only when vascular occlusions are produced by the inflammatory lesion When the pyelonephritis is unilateral, a nephrectomy is definitely to be considered, provided the affected kidney shows little if any function and the opposite kidney functions well. The urine is usually normal and the disease symptomless. The intravenous pyelogram detects the case, which then must be further studied by split renal function tests In one half of Pickering and Heptinstall's cases,[81] the blood pressure was materially reduced after nephrectomy In Rosenheim's experience,[82] arterial pressure is more apt to be reduced when the diseased kidney still shows good excretion of the dye

Fowler and I[46] reported on personal experiences with two children and one adult in whom the unilateral renal disease was eliminated by nephrectomy. All three had congenital hypoplastic kidneys In the first case, splanchnicectomy was added to nephrectomy, since the blood pressure rose after a nephrectomy done in 1942 A number of children, seen on the pediatric service of Drs Hess and Poncher at the University of Illinois, showed a uniform pattern of early hypertension, vomiting and headache in the first few years of life, which was sometimes diagnosed as brain tumor By the time the children were seen, the diastolic pressures were between 140 to 160 mm. of mercury and they were in the full-blown malignant phase with nitrogenous retention We operated on two of them, performing a nephrectomy with bilateral splanchnicectomy, but the state of the opposite kidney prevented any improvement Such patients are amenable to an actual cure if the removal of the agenetic kidney is done at the earliest possible moment, as soon as hypertension is discovered I have not seen such children lately but I suspect that the pediatrician is still unaware of the urgency of the situation, and that the persistence of hypertension may create a new mechanism which is irreversible [59]

Renal Revascularization

Ever since the emphasis of Goldblatt and his co-workers on renal ischemia in hypertension, cases of the so-called Goldblatt kidney, where the main renal artery is obstructed by an embolus or atheroma, have been looked for In spite of the previously quoted figures of Blackman,[41] stating that the arteriosclerotic plaques protruding into the renal arteries occur in 86 per cent of the postmortem examinations of essential hypertensive patients, our frequent use of aortography has visualized only a single case (fig 326). One can, however, do a splenorenal arterial anastomosis in a patient whose aortic atheroma obstructs the renal artery, or one can try renal arterial endarterectomy or graft [44 45] Since the majority of patients with essential



FIG. 326. High aortic occlusion showing left renal artery occlusion, poor renovascular pattern and increasing hypertension. (Courtesy of William S Dye, University of Illinois College of Medicine.)

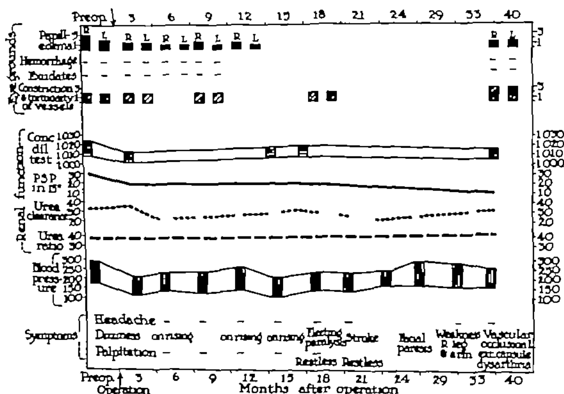


FIG 327 A bilateral nephro-omentopexy was done for Alma J., in April, 1936, in a grade 3 hypertension. She lived till 1940 with repeated small strokes, but maintained her modest renal function. The renal biopsy indicated advanced irreversible nephrosclerosis. (de Takats, G and Scupham, G W Revascularization of Ischemic Kidney Arch Surg., *41* 1394 1940)

hypertension and also those with chronic pyelonephritis have an arteriolar or at least a terminal arterial involvement, we have been interested in stimulating vascular anastomoses between omentum or muscle and the decapsulated, scarified kidney In 1940, Scupham and I[83] reported on four hypertensive patients in whom the diagnosis of a malignant phase of hypertension was made In none of these patients was there definite improvement, although one patient remained in a stationary condition for four years (fig 327) We suggested then that the cases were too far advanced, but that earlier phases of hypertension might have nephro-omentopexy Since that time, almost all our splanchnicectomized patients have a pedicled muscle flap placed in the cortical defect left by the biopsy Direct evidence on man that these pedicled muscle grafts are carrying circulation is lacking However, recent animal experiments of Baronovsky and his associates[84] show that fusion between the decapsulated kidney and a segment of small bowel from which the serosa has been removed results in vascular connections These can be visualized by an opaque substance and will permit renal function even after the main renal artery is clamped. We shall continue our interest in this mostly unexplored field

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APPENDIX 1. HYPERTENSIVE WORK-UP

ORDERS*

(Note Patient should arrive before 5 00 p m)

Date Night of admission

(1) Select, high vitamin, high protein diet
(2) 6 00 p m concentration and dilution test starts

Date First day

(1) c b c , Hgb differential and hematocrit
(2) Urinalysis
(3) Sedimentation rate
(4) Kahn
(5) Blood pressure every four hours day and night by a nurse during the entire study
(6) Blood for typing
(7) Blood chemistry BUN, N P N , cholesterol, sugar, Na, K
(8) Electrocardiogram in afternoon
(9) P S P tests in afternoon
(10) Consultation with ophthalmologist Dr
(11) Consultation with internist Dr
(12) Interview with social worker

Date Second day

(1) Urea clearance test
(2) Insulin tolerance test—*if ordered*
(3) Sodium Amytal test in p m
(4) *No* castor oil, as preparation for I V P
(5) Special tests to be done by intern in the afternoon
    (a) Cold pressor test
    (b) Postural pressor test
    (c) Tourniquet test

Date Third and fourth days

(1) Basal metabolic rate
(2) 2-M chest plate in a m
(3) Intravenous pyelogram in p m with Prostigmin (amp 1, 1 2000) preceding, instead of castor oil
(4) Etamon test
(5) Glucose tolerance test—*if ordered*
(6) Histamine test—*if ordered*
(7) Regitine test—*if ordered,* not for 48 hours after Sodium Amytal

Note Notify service *immediately* if any test is not completed according to instructions

INSTRUCTION SHEET

Concentration Dilution Test

(1) 6 00 p m to 9 00 a m No food or drink
(2) 6 00 a m Patient voids, specimen given to laboratory as "routine specimen"

* See instruction sheet for carrying out tests

(3) 6·00 a.m. to 9·00 a.m. Still no food or drink
(4) 9·00 a.m. Patient voids, specimen given to lab marked concentration specimen"
(5) 9·00 a.m. to 9.30 a.m. Patient drinks 1000 cc of water
(6) 9 30 a.m., 10·00 a.m.
10.30 a.m., 11·00 a.m.
11.30 a.m., 12·00 p.m.
12.30 p m. and 1·00 p.m
} Patient to void at these times, and specimens sent to lab marked dilution specimen #1 to #8"

P.S P Test (I V —15 minutes)

(1) Patient to empty bladder
(2) Patient to drink two glasses of water
(3) P.S P —1 cc. (1 ampule) I V., by intern
(4) Patient to empty bladder 15 minutes after injection
(5) Take entire specimen to lab

Insulin Tolerance Test

(1) Fasting blood specimen—drawn by intern
(2) Calculated amount of insulin injected I V (see below)
(3) Blood specimens drawn at 15 30 and 60 minutes after injection for blood sugar (Calculation 1/40 of a unit of regular insulin per kilogram of body weight)

Urea Clearance Test

(1) 6·00 a.m. to 9·00 a.m. Nothing by mouth
(2) 7·00 a.m. Patient to empty bladder discard this specimen patient to drink two glasses of water
(3) 7·00 a.m. to 9·00 a.m. Blood drawn by lab during this time
(4) 9·00 a.m. Patient to void specimen given to lab marked "two hour specimen for urea clearance"

Sodium Amytal Test

(1) 9:00 p.m., 10·00 p.m., 11·00 p.m. Sodium Amytal 3 gr at each time
(2) 9·00 p.m. to 9·00 a.m. Blood pressure taken every 30 minutes during this time

Glucose Tolerance Test

(1) Nothing by mouth from 7·00 p.m. the previous night until after the test is completed
(2) First a.m. urine voided is saved and marked specimen #1"
(3) Fasting blood sugar specimen drawn by intern, marked "specimen #1"
(4) Calculated amount of glucose (50 per cent dextrose) I V by intern (see below)
(5) Urine and blood specimens collected at 30 60 90 and 120 minutes and sent to lab marked accordingly (Calculation 1/3 Gm glucose per kilogram of body weight)

Etamon Test (by intern)

(1) Basal blood pressures—three
(2) Have Neo-Synephrine and hypodermic syringe at bedside
(3) Calculated amount of Etamon injected I.V in 15 seconds (see below)
(4) B P at 30 seconds, one minute and every minute for five minutes, or until blood pressure returns to pretest level
(5) Absolute bedrest for four hours after test
(Calculation 6 mg. per kilogram of body weight, and not over 5 cc.)

Cold Pressor Test (by intern or technician)

(1) Basal blood pressures—three, in quiet surroundings
(2) One arm immersed in bucket of ice water for one minute
(3) Take blood pressures at 30 seconds after immersion, upon removal and every minute for five minutes or until pressure returns to pretest level

Postural Pressor Test (by intern or technician)

(1) Basal blood pressures—three in horizontal position
(2) Blood pressure every minute for five minutes while sitting
(3) Blood pressure every minute for five minutes while standing

Regitine Test (by intern)

(1) Basal blood pressures—three
(2) 5 mg of Regitine—*intramuscularly*
(3) Blood pressures every five minutes for 20 minutes, or until lowest level is reached

Histamine Test (by intern)

(1) Basal blood pressures—three
(2) Calculated amount of histamine I V (see below)
(3) Blood pressures taken every minute for 10 minutes (Calculation. One ampule of histamine contains 1 cc of solution (1 mg) Take 0 1(1/10) cc of this and mix 1 cc with sterile saline Give 0 50—1/2 cc of this mixture No sedation for 12 hours before test No thiocyanates for four to six days Compare with cold pressor test

Tourniquet Test (by intern)

(1) Blood pressure cuff on arm with pressure midway between systolic and diastolic for five minutes
(2) Read number of petechiae in an area the size of a stethoscope diaphragm which are present 1/2 hour after cuff is removed

ACTH-Eosinophilia (by technician)

Eosinophil count is made in a special counting chamber in duplicate 25 mg of ACTH is injected subcutaneously A second eosinophil count is done four hours after the subcutaneous injection

HYPERTENSIVE WORK UP SUMMARY

Name

Eyegrounds

Two-meter chest film

Electrocardiogram

I.V Pyelogram

Urea Clearance /100 cc. blood

15 min. P.S P

Cold Pressor Test

Basal Ceiling

Time till return min.

Postural Pressor Test

Lying Standing

Sitting

Etamon Test

Basal Minimum

Histamine Test

Basal Maximum

Regitine Test

Basal Minimum

Blood Count

rbc wbc

Hgb. Hematocrit

Kahn Sed. rate

Blood Chemistry

Bl. sugar Urea N

N P.N. Cholesterol

B.M.R.

Hospital #

Date Age Sex

Urine Analysis

Color Diacetic Sp gr

Acetone pH Alb.

Sugar Casts Cells

Bacteria

Water Tolerance

AMOUNT SPEC. GRAVITY

Conc. Spec.

Spec. 1

Spec. 2

Spec. 3

Spec. 4

Spec. 5

Spec. 6

Spec. 7

Spec. 8

Sodium Amytal

Basal Minimum

Insulin Tolerance

Fasting 15 min.

30 min. 60 min.

90 min. 120 min.

ACTH-eosinopenia

Before After

Percentual drop

Tourniquet Test
(check one)

0 1 2 3 4

INTERPRETATION AND RECOMMENDATIONS

APPENDIX 2. CASE STUDY

Mrs V S , 34 years old, a married, white female ex-school teacher, was first seen by the social worker on October 20, 1953 The patient was tense and at times blocked in conversation, her chin quivered, her eyes were moist throughout much of the interview and she cried uncontrollably when we discussed her stepmother She blocked completely regarding her early development She expressed negative feelings about living in a small apartment as compared to the family's large home

Her mother was of English ancestry, born and educated in the United States (four years of high school) She died of a cerebral hemorrhage when the patient was 6 The patient spoke freely of her father, but blocked on any information regarding her mother Her father was also of English ancestry and was born and educated in the United States (four years of high school) He was described as a good provider, stubborn, quiet and unapproachable The patient was 8 when her stepmother, who was a housekeeper for two years, entered the family constellation The patient called her "mom," resenting reference to her as a stepmother and indicated that she is a warm, outgoing and active person The patient felt that her parents had some influence on the teaching career she and her only sister pursued She seemed to have rivalry with her sister, particularly in physical appearance The patient, after obtaining a degree in teaching, was unemployed for one year because of her fear to teach.

The patient met her husband at a roller skating rink and after a courtship of two years she married him Marriage taught her much about living Her husband is three years her junior, is employed as a switchman for a railroad, has a nice disposition, is dependable, responsible, unable to handle money and not very "brainy " He gives her his salary check and receives two weeks allowance from her, which he promptly loses playing cards—"I guess you would call him immature " Her interest in describing her husband seemed exhausted when she suddenly stated that he is the "damnedest liar " This brought out negative feelings that her husband's family is critical of her ability as a wife

Her husband is interested in children and for his sake she would like to have some, but she had been advised by a previous physician not to have any Her husband at first accepted this, but is again bringing up the subject Discussion of her feelings regarding the physician's recommendation brought out the fact that the patient was fearful of pregnancy because her mother had high blood pressure and died early She definitely felt that there must be some tie-up between pregnancy and high blood pressure A neighbor, who is more friendly than his wife, told her that his wife had high blood pressure and when she became pregnant the doctor told her to get rid of the child

The patient had some understanding of her present illness She had seen her present physician once and was "really thrilled" because he was so interested in her as a human being, "we all like that, don t we?"

The interview was superficial and it was felt that the patient was unconsciously withholding information in some instances She seemed emotionally immature and rigid, with much repression Besides uncontrollable tears when the subject of her stepmother and children arose, the patient's face became violently red and a rashlike flush appeared across the anterior portion of her neck, which rapidly faded away when the subjects were changed * Her comments regarding her own family and her occasional amused smiles seemed to reflect a negative relationship to all, with a rivalry with her sister She stressed the change in her cultural pattern within her physical environment, and her choice of a husband who is below her in intelligence

From October 21, 1953 to November 1, 1953, when she was discharged, the patient was seen daily (with exception of Saturday and Sunday) in the hospital Support was given as she

* An excellent description of the diencephalic syndrome

needed it. Interpretation was given carefully because of her emotional instability which continued to manifest itself in tears and quivering of her chin. These visits presented the patient s awareness of her negative feelings toward her mother in-law but she showed no insight. She apparently was in competition with the mother in-law for her husband s attention. She showed unconscious guilt in not visiting her father more often than weekends. Depressions occurred over her high blood pressure and being hospitalized. She offered service to fellow bed patients and felt rejected when they refused to accept it. She seemed unable to understand the role of a wife at times she seemed to be the mother giving her husband gifts and coddling him to get affection, and then again she seemed to be the little girl asking for attention of a good father

On January 16, 1954 the patient was seen in the Vascular Clinic. She requested the presence of her husband in the interview The doctor stated that he was considering surgery and that they had discussed it and decided that they would both feel better if she had the operation.

The patient was seen alone briefly She stated that the talks with the worker were helpful. She "feels more calm" and had been able to think through some of her problems relating to her father The annoying neighbor no longer upsets her Instead of arguing her point, the patient realizes that they are different in their thinking, and she either changes the subject or lets the neighbor talk.

She spoke freely of her father and her responsibilities to him. These were discussed and interpreted, as well as her responsibility to her husband. She frankly stated that she had guilt feelings regarding her father particularly her hostilities toward him when she recalls his many negative attitudes and behavior of the past.

She was now able to discuss more freely with her husband her feelings toward her father Her husband returned to the interview and after more discussion regarding surgery for the patient she decided definitely to make arrangements for it. In this visit the patient was able to express herself easily and unemotionally It was apparent that she was aware of her problems and that in many instances had insight.

The patient was readmitted to the hospital on January 31 1954 A left dorsolumbar sympathectomy and splanchnicectomy was performed on February 8 1954 On February 21 1954 the same type of surgery was performed on the right On the third day after the operation, the patient became depressed. With support to her feelings and with interpretation, she quickly came out of the depression. Eleven days following surgery she became emotionally disturbed as she related that a doctor had called her mother "an old witch." She was readily amenable to interpretation.

The patient stated that interviews with the social worker were helpful. She said that surgery has helped, but that there is also a need for her to understand herself and others. She became aware of her need for changing her 'attitude" toward the many things, which she expressed as "part of living." She approached her problems realistically and constructively

There were three individual visits with the husband, who seemed to understand the patient, was aware of his own problems and was realistically attempting to solve them. The husband seemed emotionally stable, and their relationship seemed to be on a more mature level. There were two interviews with the stepmother She is a comfortable person who is understanding of the patient and very fond of her There was a positive relationship among the patient, husband and stepmother The stepmother described the patient as being an unwanted child whose natural mother had never cuddled her The patient s first request of her stepmother (at age 7) was to sit on her lap and, desiring to be loved, explaining that this was never given her by her natural mother

On April 9 1954 the attending physician telephoned from his office, stated that the patient seemed quite tense because of a bladder infection and requested that the social worker assist her in seeing a urologist. Following a urological examination and treatment, the patient and husband visited with the worker The patient stated that she is certain her hypertension was not only organic, but functional. She has made adjustments which are comfortable, and she intends to keep it that way

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The patient was readmitted to the hospital on January 31 1954 A left dorsolumbar sympathectomy and splanchnicectomy was performed on February 8 1954 On February 21 1954 the same type of surgery was performed on the right. On the third day after the operation, the patient became depressed With support to her feelings and with interpretation, she quickly came out of the depression. Eleven days following surgery she became emotionally disturbed as she related that a doctor had called her mother "an old witch." She was readily amenable to interpretation.

The patient stated that interviews with the social worker were helpful. She said that surgery has helped, but that there is also a need for her to understand herself and others. She became aware of her need for changing her attitude" toward the many things, which she expressed as part of living." She approached her problems realistically and constructively

There were three individual visits with the husband who seemed to understand the patient, was aware of his own problems and was realistically attempting to solve them. The husband seemed emotionally stable, and their relationship seemed to be on a more mature level. There were two interviews with the stepmother She is a comfortable person who is understanding of the patient and very fond of her There was a positive relationship among the patient, husband and stepmother The stepmother described the patient as being an unwanted child whose natural mother had never cuddled her The patient's first request of her stepmother (at age 7) was to sit on her lap and, desiring to be loved, explaining that this was never given her by her natural mother

On April 9 1954 the attending physician telephoned from his office stated that the patient seemed quite tense because of a bladder infection and requested that the social worker assist her in seeing a urologist. Following a urological examination and treatment, the patient and husband visited with the worker The patient stated that she is certain her hypertension was not only organic, but functional. She has made adjustments which are comfortable, and she intends to keep it that way

The patient was *readmitted* on September 18, 1956, for the spontaneous delivery of a full-term female infant on September 19, 1956, weighing 5 lbs , 8½ oz She had an uneventful postpartum course and was discharged on September 24, 1956

Daily visits were made to the patient during this hospitalization She stated that her blood pressure continued to remain low during pregnancy, that it did rise during labor and delivery, but that she did not find it disturbing She stated that labor was quite comfortable, because of the dorsolumbar sympathectomy, she did not feel the contractions Both she and her husband were extremely happy about the baby They have bought a new home, the patient seemed emotionally stable and there was a positive, mature relationship between them She told of her father's death with appropriate feeling She frankly expressed that she has some anxieties about caring for the baby (this seemed justifiable since this was her first child, conceived in late years) She inquired about instructions in the care of the baby, and stated that her step-mother would help for a month after she would be discharged (September 24, 1956) She again thanked the social worker for her help, stating that both her husband and she had learned much in the visits with her which would be helpful in rearing the child The casework before and after surgery in this case was helpful, so that the patient's hospitalization portrayed her as a mature adult who has adjusted well to her environment and is able to live a meaningful and purposeful life in her community

At the last follow-up, February 10, 1958, this patient's blood pressure was 140/90 in the horizontal and 120/85 in the standing position This is a four year follow-up and is not included in our 10 to 20 year follow-up study [1]

PART IV

Surgical Technique

Happy the man whom the dangers to others make cautious

GIOVANNI MARIA LANCISI 1745

CHAPTER 20

ANCILLARY METHODS

VASCULAR SURGERY LIKE ANY OTHER TYPE OF SURGERY IS GREATLY DEpendent on methods of anesthesia hypotension and hypothermia In addition, many institutions lean heavily on artery banks, others on vascular prostheses These ancillary methods will be first briefly discussed in their relation to the surgical procedures to be described

1 ANESTHESIA

Naturally no attempts will be made to discuss the techniques of anesthesia There are, however certain peculiarities of the patient suffering from diffuse vascular disease which need to be considered in selecting the proper anesthetics or their combinations One such peculiarity is a marked sensitivity of the vasomotor center to carbon dioxide and this is especially true of hypertensive patients They will respond to small rises of alveolar carbon dioxide levels with marked hypertension and require a great deal of oxygen and controlled respiration especially in the lateral position Hypoventilation, especially in patients receiving Cyclopropane results in a restless fluctuating blood pressure curve contributing much to the detriment of the patient and to the angina of the surgeon The late Cyclopropane shock, very undesirable in vascular cases seems to be due to hypercapnia respiratory acidosis and consecutive facilitation of vagal reflexes [1] This is an excellent anesthetic, but only in excellent hands

Patients with massive blood loss, which calls for immediate replacement, whether on the battlefield or in the operating room, are very sensitive to anesthetic drugs or to excessive premedication They do far better under moderate hypothermia or in an air conditioned operating room than in a hot humid environment Any method producing sympathetic paralysis be it spinal anesthesia or hypotensive drugs may push them into peripheral circulatory failure

Patients with thromboembolic disease tolerate even small fluctuations of blood pressure very poorly While Pentothal actually lengthens coagulation time most anesthetics may accelerate clotting time and this together with a fall in blood pressure creates an ideal set up for spreading thrombosis

Diffuse arteriosclerosis makes the patient less adaptable to the stress of anesthesia Some of the tranquillizing antihistaminics, notably Phenergan in 25 to 50 mg. doses given as premedication definitely dampen the emotional and physical stress of anesthesia Demerol with scopolamine or atropine is

a useful combination Attention should be drawn, however, to the obvious fact that elderly patients with decreased liver function and diminished cerebral flow should not be subjected to a routine dose of premedication by the anesthesia resident The elderly arteriosclerotic patient who has his leg removed under spinal anesthesia may stay in a semistupor for days because of too heavy premedication During this time, his hypoventilation and bladder atony will lead to a number of complications

Spinal anesthesia has been extensively used in our clinic during sympathectomies, amputations and vein-strippings It gives excellent muscle relaxation, does not upset the metabolism and feeding program of diabetic patients and is remarkedly free of serious complications when it is performed by a single individual of the anesthesia team. Much to our regret, however, it has been necessary recently to restrict this method to anesthesia for amputations on elderly arteriosclerotic or diabetic patients The increasing incidence of postspinal headaches makes early ambulation after vein-stripping difficult, if not impossible While there has been no cord damage after many hundred lumbar sympathectomies performed since 1928 under spinal anesthesia, recently two patients have had neurologic defects with motor and sensory loss and severe paresthesia after spinal anesthesia

Whether this is the price to pay for the training of anesthesia residents or whether the simple solution of procaine crystals in the patient's own spinal fluid was preferable to stock solutions of ampules, to possible leakage of sterilizing fluids into ampules, to the changing pH of the solution or to the use of vasoconstrictors, is beyond the scope of this discussion In a short course on local anesthesia, published in 1928,[2] I made the point that the reinjection of the patient's own spinal fluid in which the anesthetic crystals are dissolved is the most physiologic method and contains no foreign substance other than procaine If the concentration of procaine is kept below 5 per cent, cord damage can hardly occur, *except in diseases of the central nervous system*, which have always been regarded as a contraindication, or if vasoconstrictors help to keep the anesthetic drug at the level of the injection

Recent large statistics, each of which contain over 10,000 cases, report a most encouraging lack of serious neurologic complications following spinal anesthesia [3] Nevertheless, the fact remains that individual cases of permanent cord damage, progressive spinal arachnoiditis and severe causalgic states are still being reported A critical evaluation of the data in the literature together with our own experience suggests that technical errors, such as inaccurate or traumatic introduction of a large needle, contamination of the spinal puncture by chemical or bacteriologic agents, errors in the concentration and composition of the drug or undiagnosed pre-existing disease of the spinal cord, are responsible for the complications Since the surgeon of today has no control over the administration of anesthesia—and fortunately so—he must have absolute faith in the team of anesthetists before he subjects his patients to quite infrequent but spectacular hazards Repeated spinal anesthesias, as used in two stage lumbar sympathectomies done a week apart, seem to increase such complications.

Certain methods of local anesthesia for diagnostic purposes such as a block of somatic and sympathetic nerves have already been described in part II (pp 67–78) of this volume Here I wish to emphasize the advisability of infiltrating the sympathetic chain and ganglia with procaine prior to their section In sympathectomies this will prevent vasospasm following section or clipping of the trunk [4] In splanchnicectomies the potent sensory impulses which may affect cardiac and other visceral impulses are blocked In amputations the infiltration of the sciatic nerve abolishes the occasional hypotension seen under light general anesthesia, but not under spinal anesthesia It is a good habit to acquire and though often superfluous it is occasionally very useful

2. CONTROLLED HYPOTENSION

Curiously enough, surgeons used to dread a fall in blood pressure during operations I well remember Professor Arcé the famous Argentine surgeon and later Dean of the University of Buenos Aires perform a cholecystectomy for us at the Research and Educational Hospitals of the University of Illinois He did his own spinal anesthesia, placed the patient in a Fowler s position and did a rapid cholecystectomy in a completely bloodless field The patient s systolic blood pressure fell to around 80 mm. of mercury, and the diastolic pressure could not be measured Arcé said at the time (1935) that this was a harmless procedure and that he had seen no harm from it Most of us were flabbergasted at this performance

The systematic use of hypotension to control bleeding and shorten the operative procedure has been started by arteriotomy approximating hemorrhagic shock then preganglionic sympathetic blockade was done by inducing high spinal anesthesia Finally sympathetic blocking agents, first hexamethonium and later Arfonad, have been employed together with the postural effect of the reverse Trendelenburg (head up) position Hampton and Little[5] have outlined the historical development of these techniques and reported the results of a questionnaire filled out by 144 anesthesiologists who used controlled hypotension on 6 805 patients As might be expected the complications encountered consisted of renal cerebral and coronary insufficiencies cardiac arrest and reactionary hemorrhage Their statistics clearly indicated that 80 mm of mercury is the critical level of blood pressure above this complications were rare However the rate of fall the prehypotensive level of pressure and the state of the vascular tree must be important factors

While the cerebral blood flow in the unanesthetized patient remains remarkably constant after an average reduction of 44 per cent in mean arterial pressure because of a corresponding decrease in cerebrovascular resistance [6] it is not to be supposed that patients with cerebral arteriosclerosis and under the influence of premedication anesthetic agents anoxia and alterations in CO_2 tension and pH—all of which occur under general anesthesia—would be capable of such compensatory response

Hypotensive anesthesia does reduce the amount of blood lost during

major operative procedures, but it does not shorten the operative time.[7] It is difficult to determine whether such reduction is sufficient to justify the hazard involved In vascular surgery, however, the clamping of a brittle atheromatous aorta is safer under controlled hypotension with about 100 mg of Arfonad used in conjunction with hypothermia This drug, as will be brought out in the next chapter (p 592), may actually make hypothermia safer by decreasing myocardial irritability and by decreasing peripheral resistance in the visceral organs, which can thus maintain a better blood flow. Certainly the method requires much experience and skill In unskilled hands it is definitely dangerous

3 HYPOTHERMIA

Reports emanating from French authors on artificial hibernation stressed the point that an experimental animal and also the human body "readily submit" to an aggression by cooling when some autonomic reflexes, which would tend to counteract general body cooling, are inhibited [8] Thus evolved the "lytic cocktail" consisting of Thorazine and Phenergan, which in themselves lower blood pressure and slightly cool the body Since cooling the body below 84° F greatly enhances cardiac irritability, the desirable temperatures are between 86° and 90° F In this state, the premedication with Thorazine and Phenergan will also maintain a moderate hypotension Combined hypotension and hypothermia have become important adjuncts in cardiovascular surgery, especially in open heart surgery requiring a dry field, and in thoracic aneurysms Whether clamping the aorta below the diaphragm for a reasonable amount of time (60 to 90 minutes) requires hypothermia or not is a debatable question, since many such operations are being performed safely without it The method is comparatively new and it is likely that its side effects will be more and more eliminated Bypass procedures may further restrict its use.

Physiologic Considerations

In their winter sleep the "true hibernants," such as the European marmot, the American groundhog and some of the ground squirrels, become poikilothermic, their deep body temperature being only a degree or two above the temperature of the environment.[9] Though most reflex activity and irritability is depressed in the hibernating animal, there remains in him a protective reflex of great interest When the environmental temperature drops below a certain level, the groundhog emerges from his burrow and rewarms himself

Man, of course, has no such protective reflex, and when he is cooled down to below 86° F (30° C) and his adaptation to the stress of cold is inhibited by Thorazine and Phenergan, he is subject to danger from essentially two sources Cardiac arrhythmia develops, notably ventricular fibrillation and standstill, and the clotting mechanism is disturbed, first, when the bleeding time is prolonged with a decrease in platelets and later when the clotting time is prolonged, thus producing hemorrhage. Most importantly,

oxygen consumption is decreased to about 55 per cent at 86 F so that arterial occlusions to the brain, heart kidney and spinal cord will be tolerated for a longer time without irreversible damage

The literature which has developed in the last few years on hypothermic anesthesia is voluminous and there is no need to cover it here Bigelow[10] Bailey[11] and Swan[12] have contributed along with many others, to our knowl edge Hypothermic anesthesia is an effective tool but a dangerous one if not in skillful hands Ventricular fibrillation has been prevented or abolished by potassium, by neostigmine by acetylcholine and by 5 per cent carbon dioxide with oxygen It is the general consensus of opinion that a cold heart is much more difficult to defibrillate than a warm one

Selection of Patients for Hypothermia

In this monograph cardiac and thoracic vascular lesions have not been discussed, and obviously it is here where hypothermic anesthesia has its greatest usefulness Since clamping of the abdominal aorta below the renal arteries may result in hemiplegia or in extensive distal thrombosis hypothermia has been in use especially when the clamp was employed for arterial grafting. On the other hand, removal of an aortic embolus would not necessitate hypothermia because of the relatively short time an occlusive clamp is in place Anesthesiologists like to use hypothermia when equipment is avail able, since very little and light anesthesia is necessary and since the patient s state, manifested by pulse blood pressure and temperature is very stable Nevertheless, the inherent hazards of the method are, in my opinion such that it only should be used when prolonged clamping of large arteries espe cially above the diaphragm is planned and when the decrease in oxygen consumption is important.

Hypothermic anesthesia is indicated only (1) when the operating room team can recognize and cope with cardiac standstill or ventricular fibrilla tion (2) when occlusion of the entire circulation is required to perform intracardiac surgery, (3) for cyanotic patients whose oxygen demand should be reduced and (4) when regional occlusion of the circulation is desired for prolonged periods Hypothermia should not be used simply to produce hypotension since the "lytic cocktail" alone or a ganglionic depressant such as Arfonad is entirely sufficient Temporary shunts may eliminate much hypothermia

Technique

In most instances—and, of course individual variations are frequent—the patient receives 50 mg. of Demerol and 0 4 mg of scopolamine three quarters of an hour before entering the operating room. On his arrival in the operating room, he receives intravenously 25 to 50 mg. of Phenergan, diluted in 10 per cent physiologic saline solution.

When the blood pressure is low Thorazine may just as well be omitted in case of hypertension, it can be given intravenously in 5 to 10 mg doses every 10 to 15 minutes thus avoiding a single large dose of 50 mg. as the

French authors[8] described it. Prechilled intravenous 5 per cent dextrose is started in a leg vein with a 15 to 16 gauge needle. Anesthesia is started with the intravenous injection of 2½ per cent Penthothal in 20 cc of normal saline With a second syringe 5 to 7½ mg of dimethyl tubocurarine is injected in 10 cc of normal saline. Oxygen is given with full control of respiration When relaxation is adequate and the patient's color is good and his pulse is slow, intubation is carried out with a cuffed tube A second intravenous infusion is started and the patient is slowly and carefully moved on to the cooling blanket. A second blanket is placed on top of him and a third blanket surrounds his head At this time, the thermostat is set to 32° F and the patient is gradually cooled down to 93° and then to 90° F., the procedure taking usually from 40 to 60 minutes

Ether is given in small quantities, but seldom is the vaporizer used One mg doses of curare are given from time to time, if more relaxation is desired

The patient's temperature is controlled by a thermocouple placed in the rectum, but a mercury thermometer passed through the nose may be a good substitute. Speeding up of the intravenous dextrose or blood may help to depress the temperature, but when this is done too rapidly ventricular fibrillation may occur

If clamping of the aorta is about to take place, the temperature is optimal at 86° F , not lower The wandering of the temperature below the desired level must be watched for, since it may drop as much as 4° F , unless one quickly reverses the temperature in the blanket before optimal cooling is reached, probably as it reaches the level of 89° F

Clamping of the vessel, especially of a brittle, arteriosclerotic aorta, requires hypotension and this may be rapidly obtained by an intravenous dose of Arfonad Blood, of course, must be replaced as it is lost, both the surgeon and the anesthesiologist learn to estimate the blood loss and must promptly replace it

After the operation is completed, the patient may be left in the operating room, wrapped in the blankets to raise his temperature He may also be warmed in his room with the electric blanket or carefully applied hot water bottles Should shivering occur, barbiturates or Thorazine, in small doses, will control it Oxygen is continued by catheter, mask or tent and the usual array of postoperative management is employed Narcotics now are kept at a minimum

This brief description is simply a bare outline of the involved procedure requiring attention, skill and experience The recent tendency of our anesthetists has been to use moderate hypothermia (86 to 90° F) and to use small intermittent doses of Phenergan and curare with less and less Thorazine and little Arfonad, thus making the procedure safer and more flexible

SIDE EFFECTS

Basically, the cardiac arrhythmias and the changes in the clotting mechanism are the two important areas to watch

In animal experiments as well as human continuous tracings of the electrocardiogram can be made using lead II or the unipolar lead aV_F

A recently developed cardiac monitor gives a simple visible and audible control of the rate and rhythm of the heart and readily differentiates cardiac arrest and ventricular fibrillation from peripheral collapse [13] It can be gener ally stated that cardiac arrest is far preferable to ventricular fibrillation and, in fact, may be purposely induced by potassium neostigmine or acetylcholine[14] to permit a "silent heart during intracardiac surgery Cardiac arrest is much more readily reversible to normal rhythm than is ventricular fibrilla tion in fact, defibrillation usually induces cardiac arrest from which state the normal rhythm can be restarted The recent thought that 5 per cent carbon dioxide in oxygen as a breathing mixture will prevent arrhythmias during hypothermia deserves serious consideration and has had some confirmation in humans [15] This however is contrary to the general opinion that continu ous hyperventilation is important in preventing an accumulation of carbon dioxide to occur since the carbon dioxide greatly increases the incidence of ventricular fibrillation [16] It seems likely that hypoxia is the basic cause of arrhythmia whether it follows hypoventilation of alkalosis following hyper ventilation. Hypoxia with 5 per cent carbon dioxide seems to be better tolerated a statement made long ago by our former colleague at the Univer sity of Illinois Ernest Gellhorn [17]

It is to be expected that increasing safety measures will be developed in the control of cardiac standstill and ventricular fibrillation. One thing is certain rapid cooling and cooling below 86 F (30 C) increases the in cidence of ventricular fibrillation Of course temperatures down to 90° F (32 C) can be accomplished by icebags to the axillae and groins by Thora zine and by a cool operating room and they may be useful in certain types of vascular surgery not requiring an open heart or in prolonged clamping of the thoracic aorta The use of Arfonad (trimethaphen camphorsulphonate) has been advocated to decrease myocardial irritability during cooling.[18]

Changes in the *clotting mechanism* have also received some attention, but the picture here is confusing and contains contradictory findings Many investigators have reported on the prolongation of clotting times Delorme spoke of the anticoagulant effect of hypothermia "[19] but most of these studies have been made on dogs whose clotting mechanism notoriously differs from man s Most surgeons including ourselves have had the impres sion of diffuse oozing during surgery under hypothermia, but there are so many other factors at play such as premedication the anesthesia, depressed ventilation and multiple transfusions, that carefully controlled experiments are desirable Prolonged bleeding time and lowered platelet counts have been found under well controlled conditions but these were promptly reversed on rewarming the experimental animal.[18, 19] Other factors in the clotting mecha nism did not seem to be disturbed Helmsworth and his associates empha sized the low platelet counts and poor clot retraction [20] This may well be a response to stress, although cooling, at least by immersion does not provoke a stress response as measured by peripheral 17 hydroxycorticosteroids On

the contrary, hypothermia with a concomitant reduction in body metabolism simultaneously depresses production and conjugation of the steroid hormones Stress response is actually inhibited under hypothermia,[21] and whether this is desirable or not is unknown at present

EVALUATION OF THE METHODS FOR COOLING

There are at least three techniques of cooling the patient. Immersion into ice water, which Henry Swan uses on children,[12] is inexpensive and rapid It seems drastic and perhaps messy Cooling by rubber blankets is probably the most widely used method at present Adults can be cooled to 80° F (30° C) in one and a half to two hours through an expensive instrument which does go out of order Cooling may also be accomplished by perfusing the pleura or the peritoneum, or by venous cooling, which consists in removing the blood, refrigerating it in coils and returning it to the inferior vena cava through the saphenous vein.[22] It takes 20 to 30 minutes to cool a patient to 30° C. but, of course, this can only be done when an open thoracotomy is present. Rapid rewarming is advocated by most experts in the field, especially if there is cardiac arrhythmia, although the danger of rapid rewarming versus slow rewarming has not been adequately investigated

Since this monograph does not deal with intracardiac surgery and not even with operations on the thoracic aorta, the field of hypothermia for our purposes is quite limited Resections of the abdominal aorta necessitating a clamp for 30 to 60 minutes are ordinarily well tolerated, although paraplegia from spinal cord ischemia has been reported Thus temperatures below 32° C are hardly ever necessary, and in only one such case have I observed cardiac standstill.

The dangers of hypothermia must be balanced against its advantages, and in peripheral vascular surgery there is only occasional need for it

4. THE ARTERY BANK

The purpose of an artery bank, like any other tissue bank, is to collect, sterilize, store and distribute arterial segments which can be safely implanted into the human body

At this time there are a number of artery banks throughout the country. The Municipal Artery Bank in Chicago has greatly profited by the experience of the tissue bank at the National Naval Medical Center and by frequent personal communications with Charles Hufnagel, Michael DeBakey, Oscar Creech, Jr , R A. Deterling, Jr. and Travis Winsor, all of whom, with many others, have been pioneers in this field A report of the Committee on Blood Vessel Banks under the Chairmanship of Jere W. Lord, Jr., also became available [23] Our bank, which had two sister banks at Northwestern University and Children's Memorial Hospital, required a somewhat different set-up, since the purpose here was to furnish reliable, readily stored arteries

to all surgeons of the metropolitan area of Chicago and to all hospitals in which major vascular surgery could be performed

Attempts were first made to base this city wide bank on the Artery Bank of the Research and Educational Hospitals of the University of Illinois which was the first bank to operate in this area It became obvious that the University could not house and provide bookkeeping for a municipal project Through conversations initiated by Dr Lester R Dragstedt with Dr Hans Popper of the Department of Pathology of Cook County Hospital, the extensive autopsy material of Cook County Hospital was made available for the procurement of arterial segments While other hospitals contribute to our source of material, the bulk of segments originate from here feeding the central bank

Early in our efforts to supply the community with frozen human arteries for implantation, the thought occurred to the Advisory Committee of the Chicago Artery Bank * that small decentralized banks might be established and built around surgeons who are doing the major part of vascular surgery In addition to the bank at the University of Illinois which functions under a small committee, we suggested banks at Children s Memorial Hospital, at Northwestern University and at a few other hospitals offering them a small initial priming dose of funds Of these the groups at Children s Memorial and Northwestern blossomed out, and their great service to those in need of such vascular transplants is outlined on page 548 They have recently discontinued their operations so that the central bank now processes all the human material

It became obvious that the majority of hospitals, either because of a lack of sufficient autopsy material or because of insufficient funds, would not be able to support such local banks Also a centralized operation could be better standardized and supervised For this reason after considerable birth pains consultations and drawing up of safeguards the central bank was born and after a short testing period began its service on July 1 1955

Procuring Arterial Segments

The vessels are obtained at a routine autopsy from subjects under the age of 40 who show no disease of the aorta and are free of infectious diseases collagen diseases or tumors in the area of the vessel A scrupulous selection of the proper type of material is carried out by a thoroughly indoctrinated pathology resident who is a salaried member of the artery bank The vessels must be obtained not later than six hours after death if the body has been immediately refrigerated after death this limit can be extended to 24 hours The removal of these vessels is done with every effort to preserve their integrity and to leave the branching arteries long so that they can be ligated easily just before use Blood is obtained for testing for syphilis and the vessel is immediately placed in a flat dish and freed from surrounding fat and loose tissue

* This committee now is functioning as the Committee of Cardiovascular Surgery of the Chicago Heart Association with myself as Chairman.

Sterilization of Arterial Segments

Most of the early banks set up regular operating room procedures to remove the arterial segments with aseptic technique This requires two surgeons and a nurse, who must be available at any time of the day and night One of the members of the Department of Surgery at the University of Illinois, Dr William S Grove, spent many evenings and nights at various morgues obtaining suitable specimens All this difficulty was obviated by the discovery that such segments can be removed without any sterile precautions and then exposed to sterilizing agents which reliably destroy bacteria and viruses without injuring the vessel wall Our bank has used two of these agents, ethylene oxide and betapropiolactone Clinical observations, however, recently reinforced by an experimental investigation by a member of our committee, Dr O C Julian, have shown that, while its sterilizing action is reliable, betapropiolactone as used in our bank does weaken the wall of the vessel even after careful neutralization [24] Segments placed into dogs did not heal nearly as well as when ethylene oxide was used While betapropiolactone is inexpensive and not inflammable, we have discontinued its use. Other banks, however, including those used by R A Deterling, Jr., in New York and D E Szilagyi in Detroit have endorsed this agent.

Our present sterilizing agent is ethylene oxide, which has to be used under a hood or in a fire-proof cubicle The technician must also carefully avoid inhaling any of its fumes or spilling it on the skin After the liquid ethylene oxide has been in contact with the blood vessel segment for 30 minutes, it is decanted and the vessel is rinsed to eliminate any trace of the agent and transferred to a sterile tube This ends the second stage of the procedure and at this time bacteriologic cultures are made for aerobic and anaerobic bacteria, and a small segment of the vessel is taken for histologic study

Ethylene oxide may not be the final answer to sterilization Four per cent formalin has been tried, but such a method may lead to premature calcification in the transplant 70 per cent alcohol has been used both experimentally and clinically, and one bank uses penicillin-streptomycin solution Since we are in mass production we cannot change our procedure until we are sure that it improves end results

Sterilization can also be effected by high voltage cathode ray irradiation, such as that used by J G Trump at the Massachusetts Institute of Technology 2 5 million roentgen equivalent physical units (rep) are used, or gamma ray irradiation can be employed using cobalt tubes, administering 2 to 3 million roentgen equivalent physical units Both methods require expensive equipment and to date we have not used them in Chicago There is also a possibility that such radiation may damage the protein molecules of the vessel wall

Preservation of Arterial Segments

Immediately after sterilization the vessel, contained in a Pyrex tube up to 50 cm. in length, is submerged in a dry ice acetone bath at −78 C. and then placed in a deep freeze unit. This rapid freezing minimizes both the size of crystals as they form in the tissues and the denaturing of proteins The vessel is kept at this temperature until lyophilization

The quickly frozen vessels can be maintained for a period of several months However, for simple storage at room temperature the dry freezing process widely used in the food industry is an excellent procedure and such a dry freezing unit has been established at our central bank The lyophiliza tion is carried out under a vacuum of approximately 1 η (0 001 mm) Hg for a total drying time of 8 to 10 hours Upon completion of the drying process the glass tubes are sealed with an oxygen torch while still under vacuum The tube is constructed with a nipple like side arm At the time the artery is to be used this side arm is broken off and the vessel is immersed in warm physi ologic saline solution for 30 minutes It thus regains its pliability and has an almost normal consistency

According to present experience these sealed vacuum tubes can be kept at room temperature for at least two years and many of them are in the instrument cabinets of our local hospitals ready to be used at any time

Distribution of Arterial Segments

When the surgeon requests a suitable artery from the bank it is de livered to him by the American Red Cross In an emergency this can be done day and night, but most of the time a 24 hour notice is desirable The bank keeps a file concerning the type of vessel when and where it is obtained to whom it is delivered and what the nature of the operation is Forms go out with each vessel * The first form is for the information of the surgeon giving a description of the vessel with measurements and instructions on how to break the tube and handle the vessel before implanting it. The second form is a questionnaire for a description of the operation and a follow up stating the results obtained

With the help of a special consultant, Dr Oscar Creech Jr then at Baylor and now Professor of Surgery at Tulane University, we have obtained a smooth, well supervised organization The accompanying table shows the performance of the Central Bank and its sister banks during its first two years of operation from July 1955 to July 1957 (Table XVI) We are now in the process of accumulating data as to how the transplanted segments func tioned Such a report is essential to tell us whether this elaborate set up which originally cost the Chicago Heart Association approximately $8 000

* All forms are shown in the appendix, pp 553–556

Table XVI

ANNUAL REPORT OF THE ARTERY BANKS* OF CHICAGO
JULY, 1955 TO JULY, 1956

	PROCESSED	DISBURSED	NUMBER OF HOSPITALS	SURGEONS	IMPLANTED
Central Bank	312	267	16	20	85
N U Bank	96	55	5	8	40
Children's Memorial	91	23	2	4	20
Total	499	345	23	32	145

* In the Second Annual Report, covering data from July 1, 1956 to June 30, 1957, 229 arterial segments were processed including 80 abdominal aortas, 52 aortic arches, 62 thoracic segments, 7 femoral segments, 9 whole infant aortas and 19 short aortic or arterial segments 40 more grafts were placed than the year before and 7 additional surgeons and 8 additional hospitals participated (From the Annual Report of Milton Weinberg, Assistant Medical Director, Central Artery Bank of the Chicago Heart Association)

and now costs it $12,000 a year, should be maintained or whether simpler procedures, such as the use of plastic tubes, are equally efficient

5. VASCULAR PROSTHESES

A historical survey of the accumulation of knowledge leading to the replacement of injured, thrombosed, inflamed or degenerated blood vessels is rewarding It took Alexis Carrel's experimental studies about 50 years to bear fruit [25] Perhaps the most potent stimulus for the spectacular advances in this field is the development of surgical laboratories in which new materials are continuously being tested Correlations between success in the experimental animal and in the arteriosclerotic man, however, do not always exist and the most carefully tested synthetic material may fail in the human [26] It should be remembered that Robert Gross in 1946 and Charles Rob in 1950 implanted frozen human arterial segments which are still functioning [26 27]

The tendency today is to lean favorably toward synthetic prostheses, and only two factors, *i e*, experience in numbers and the time elapsed since implantation, will solve the problem of the best substitute for a closed or aneurysmal segment Obviously, however, graft failure should not always be levied against the implant when progressive arterial disease above or below the transplant is responsible

This area of vascular surgery is advancing so fast that any definite statement made today may have to be revised tomorrow. However, certain observations may be cited, which tend to show the dynamic advancement of ideas The line of progress, which is true of many other surgical endeavors, is not a straight ascent but is somewhat jerky, with some initial enthusiasm having to be dampened by a careful and long enough follow-up of at least two but preferably five years' duration

On the basis of a review of the literature and of experience in our own institution, the following points seem to emerge

(1) Nothing is more desirable than fresh, autogenous material the minute the autogenous material is dry frozen the percentage of failures rises [28]

(2) Homologous dry frozen veins placed in the thigh as a bypass show a large percentage of elongation tortuosity and aneurysm formation in man This material is easily obtained during vein stripping, but its use has been abandoned.[29a] An autogenous vein however is desirable material [29b]

(3) Degenerative changes in transplanted aortic and femoral lyophilized homografts, as studied by serial angiographic findings in 150 cases and correlated with the histologic study of 8 aortoiliac and 15 femoral graft specimens led Szilagyi and his co-workers[30] to the conclusion that both the human aorta and the human femoral artery are poor arterial substitutes The femoral artery since it is a muscular and not an elastic artery is particularly prone to degenerate Progressive degenerative changes at the suture line are most disturbing in the femoropopliteal area, where they may lead to late closure

(4) At the present writing, the emphasis is on plastic substitutes notably crimped, two-way stretch knitted, woven or braided material Starting with the pioneer material of Blakemore, Jaretzki and Voorhees [31] interest today is focused on Dacron Orlon and Teflon all of which seem to retain their strength when implanted into living tissue In an excellent chapter on unsolved problems in his monograph on plastic arterial grafts Wm Sterling Edwards[32] makes the point that one of the qualities of the ideal graft must be a long term preservation of tensile strength but that the heavy braided Nylon may not be inferior to Dacron or Teflon

(5) The argument whether porous or nonporous material is preferable is still not definitely settled, although much evidence favors porous material While there is bleeding through these pores so that the material needs to be preclotted one hopes that fibroblasts and capillaries will grow through the interstices of the plastic material and provide greater vascularity for the neointima and more strength for the final tubular structure The histologic studies of Szilagyi and his co-workers [30] however would indicate that as far as the frozen homografts are concerned a true replacement of the graft by the host does not occur and when a long enough period has elapsed the reaction of the host toward the plastic porous prostheses supposedly used as a scaffold, will also have to be examined The optimal prosthesis then will be one that retains its tensile strength for many years and is gradually incorporated into the host. It is likely that the torrential implantation of various newer prostheses will begin to show some untoward reactions in the future including possible carcinogenesis, and that a sober reassessment of these materials will then ensue [33]

(6) While several clinics have given up frozen homologous arterial grafts entirely except for some specific locations with multiple branches such as the celiac axis or the aortic arch, the abandonment of artery banks espe

cially if they are well functioning, would be premature and is not contemplated It is true, however, that many institutions and hospitals have a difficult time obtaining cadaver material and this, together with the ease of sterilization, indefinite shelf life and ready adaptability to all lengths and diameters, is a potent argument for plastic prostheses

(7) The recent report on the clinical use of synthetic arterial substitutes by Crawford, DeBakey and Cooley[34] favors a flexible knitted Dacron tube, which they have used without a single "graft failure" in 237 cases in all segments of the aorta and peripheral arteries, including those requiring multiple branches such as the aortic arch and the upper abdominal aorta Other flexible, crimped, two-way stretch material, such as the Helanca seamless tubes used by Szilagyi, the crimped Dacron tube developed by Julian in our institution and the pioneer crimped material of Edwards and Tapp, all have their adherents and are probably equally efficient in experienced hands They do vary in porosity and thus in the initial loss of blood oozing through them, in consistency and in the amount of fraying when cut DeBakey's crimped Dacron tube does not fray when cut, but heat sealing with a cautery is an easy matter Teflon is favored by Harrison

In my limited observations, these grafts are perhaps a little more difficult to handle, although this is no doubt the kind of statement surgeons first made about the use of rubber gloves More important is the percentage of leaks at the suture line and the incidence of infection which, while no accurate figures are available, seems to be higher than with the arterial homograft

(8) The last word on heterologous grafts taken from the calf, sheep, pig or horse has not yet been said Obviously one runs into difficulty with an antibody response of the host Medawar[35] has listed a number of methods to inhibit antigenicity, but all of these seem to impair the reactivity and the wound healing of the host Culturing the heterologous graft in the donor's own serum to inactivate the antigenicity of the foreign protein has been done in the case of "cultured" bone and has been proposed for blood vessels No series of patients having such grafts implanted have come to my attention so far

Another approach in which enzyme-digested bovine carotid arteries have been sterilized and stored in propylene glycol has had experimental trial[36] These flexible, though inelastic, nonporous tubes consist mostly of collagen fibers, the elastica having been digested, with this the foreign protein is said to have lost its antigenicity In animal experiments, these materials degenerate, but in unpublished observations with Ian Duncan Thompson, we have observed a certain percentage of takes No adequate clinical trial has been given this material on our service

A tremendous literature has grown up around the subject of arterial substitutes and continuous improvement in case selection and in the choice of material is bound to ensue Our laboratory has experimented with steel mesh, fiberglas and a second type of collagen tube, these were originally used to learn about tissue reaction to implantation and were not employed clinically[37] For a thorough discussion of the subject together with criteria

for the suitability of synthetic materials implanted into mammalian tissue the report of the Committee on Vascular Prostheses of the Society of Vascular Surgery under the Chairmanship of Oscar Creech Jr may be consulted [38] Of a total of 256 collected cases with vascular substitutes 93 per cent have been successful a favorable comparison with the results obtained with homografts I agree however with Deterling[39] that, at least to date arterial homografts have given as good if not better end results than synthetic materials Time will show

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APPENDIX 1 FORMS FOR THE DISTRIBUTION OF ARTERIAL SEGMENTS

Hospital

Prosector

Case number

Age of donor yrs. mo Sex male female

Height ft. inches

Weight pounds

Cause of death

	time	date
Death	a.m. p.m	
Refrigeration	a.m. p.m.	
Autopsy	a.m. p.m.	
Sterilization	a.m. p.m	

Sterilization method B.P.L. eth. ox.

Kahn Bacteriology

Lyophilization date

Graft number

Released to date

Form returned date

ARTERY BANK
CHICAGO HEART ASSOCIATION
637 South Wood Street
Room 317
Chicago 12, Illinois

Abdominal Bifurcation

Graft number

Date obtained

To sterilize the tube, immerse it in Zephiran 1 1000, or its equivalent, 45 to 60 minutes

To open the tube and rehydrate the vessel, immerse the top of the tube in sterile 0 85 per cent saline, so that the side arm is under the fluid level and the vessel is not opposite the side arm Knock off the side arm with a sharp, quick blow The tube will fill with saline Right the tube and knock off its top at the scratch Let the vessel stand in the solution 20 to 30 minutes, it is then ready for use

Inrush of fluid *must not* strike the vessel

Note All observed defects are marked on the diagram in red ink Wet dimensions

ARTERY BANK
CHICAGO HEART ASSOCIATION
637 South Wood Street
Room 317
Chicago 12, Illinois

Telephone MOnroe 6-9787 (bank)
MOnroe 6-2250 (technician s home number)

Date of report

Graft number

Date graft obtained

Recipient patient s hospital number

Date of operation

Hospital

Diagnosis

Type of operation

Condition of patient at time of report

(Signature of surgeon)

Note Prompt completion and return of this form is absolutely necessary

CHICAGO HEART ASSOCIATION
69 West Washington Street
Chicago 2, Illinois

ARTERY BANK
CHICAGO HEART ASSOCIATION
637 South Wood Street
Room 317
Chicago 12, Illinois

Dear Doctor Grove

On (date) we released to you arterial segment(s)

number(ed)

You reported the kind of surgery as

performed on (date), on (patient's name)

aged , (hospital number), at (name of hospital)

In order to complete our records will you please check the answers to the question listed below and send this letter by return mail? Thank you

Sincerely,

Medical Director

Is the graft patent or closed? patent ( ) closed ( )

If closed, how soon after the operation did it close?

Did any complications develop?

M D
(Signature)

Date

CHAPTER 21

SURGICAL PROCEDURES

THIS PART OF THE MONOGRAPH IS NOT TO BE CONSTRUED AS BEING A SURGICAL atlas There are a number of excellent descriptions of how to approach suture anastomose and bypass certain vascular segments of the body Rather in this chapter I wish to illustrate with the help of simple black and white line drawings the principles of technique The actual procedures, in my opinion have to be learned in the experimental laboratory and in the operating room and not in movies, telecasts or panels stimulating as they are to those who already have had experience The experimental surgical laboratory has given a wonderful opportunity to the young surgeon to investigate a method to test a vascular substitute and at the same time to perfect his skill in doing vascular anastomoses on small vessels often harder to do in animals than in humans

INSTRUMENTS AND SUTURE MATERIAL

It is natural that with new developments in vascular surgery instruments had to be developed to facilitate these procedures Figures 328 and 329 show the most common instruments employed on our service Obviously these are just a few examples a vast set of new instruments has been devised, but they can usually be boiled down to a few common denominators In spite of the facility with which the arterial current can be interrupted by the placement of noncrushing arterial clamps, attention should be called to the clamp injuries caused in sclerotic vessels below the level of the iliac arteries The experimental findings of Taylor and his associates[1] have a bearing on this subject since they showed that localized injury such as freezing, favors the deposition of cholesterol in rabbits made atherosclerotic by cholesterol administration Clamp injury has received little mention in recent times but more and more the return to rubber tubing and cord tapes will become prevalent as intramural hemorrhage and late stenoses of clamped arteries are being recognized Especially are the distal clamps on femoropopliteal segments and the clamps on collaterals during endarterectomy preferably done with rubber tubing The late effects of prolonged clamping on sclerotic segments is difficult to evaluate but may need serious re-examination

Suture material in vascular repair is usually 00000 Deknatel silk but in the atherosclerotic aorta 000 silk is far more preferable Number 00000 Dacron (Mersilene) sutures have also been used The tensile strength of the Mersilene suture is excellent, and it even shows some distensibility which is advantageous in a pulsating vessel However its diameter is a little too large and possibly a smaller sized needle would be preferable

All needles are swaged to the sutures, and the design of adequate needle holders to carry these fine needles still needs further refinement Most surgeons, including myself, prefer diamond-jaw needle holders, but some thought is being given to a needle holder with a slot in its jaw; however, this will prevent the placement of the needle at an angle Straight needles and double-pointed needles are also favored by some surgeons [2] The best needle and suture, whether colored or white, is the one to which the surgeon is accustomed and which his instrument nurse prefers, should one be lucky enough to have an instrument nurse as a permanent member of the team.

FIG 328 Instruments used in lumbar sympathectomy· (A) a head lamp facilitating the visualization of the crus of the diaphragm from the retroperitoneal approach, (B) Crile dissectors used for picking up and dissecting the sympathetic chain; (C) a small artery dissector (Harriet); (D) a ligature ball containing a spool of 100 cotton or 00000 Dacron (Mersilene). (E) a Cushing clip holder especially made 9 inches long with a clip carrier. and (F) a Deaver retractor Of these four are sterilized.

For transthoracic sympathectomies, a Finochietto ribspreader and instruments for rib resection are added

FIG 329. Instruments used in direct vascular surgery. (A) Small, straight and curved bulldog clamps with rat tooth blades, (B) a Carrel clamp; (C) two right angle Southwick clamps. (D) a long diamond-jaw needle holder. (E) a plastic tube, 065 (Becton and Dickinson), threaded on a no 20 gauge needle for intra-arterial injections. (F) two aortic clamps; and (G) the set of arterial strippers devised by Cannon and Barker for endarterectomy. The instruments shown are DeBakey's bulldog and aortic clamps made by Pilling, Southwick's clamps made by Richter, and Cannon's arterial stripper made by Surgical Instruments Service, Los Angeles, California

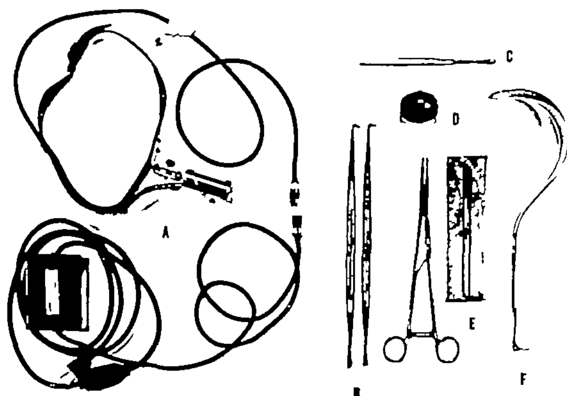


FIG 328 (*See opposite page for legend*)

FIG 329 (*See opposite page for legend.*)

EXPOSURE OF MAJOR ARTERIES*

Figure 330. The Carotid Bifurcation

A Place the patient's head reclining with his neck turned, but not overextended, toward the opposite side.

B Incise transversely at the level of the cricoid cartilage, centering over the palpable carotid pulse. Divide the skin, subcutaneous fat, platysma and fascia in the same direction

C Expose the anterior border of the sternocleidomastoid muscle Retract this muscle laterally, the superior belly of the omohyoid forms the inner border of the triangle in which the neurovascular bundle lies

D Within the vascular sheath the descending branch of the hypoglossal nerve lies anteriorly, the internal jugular vein is next, and the common carotid artery is slightly behind and medial to the vein.

E When tying the large vessels do not pick up the vagus nerve which lies closely behind them The sympathetic chain lies outside of the vascular sheath. The common facial vein, entering the internal jugular at the carotid bifurcation, may have to be tied for better exposure.

F. When the common carotid artery is tied, collateral circulation is established through the vertebral and the opposite internal carotid, and retrograde through the external carotid Avoid trauma to the bifurcation, use procaine to block the nerve receptors in the carotid sinus. Suture or graft is always to be attempted

UNFAVORABLE EFFECTS If the artery is ligated or if it becomes occluded, permanent cerebral damage may result To minimize such damage, manual compression of the pulsating vessel tests for collateral circulation, especially the integrity of the circle of Willis and the vertebral arteries Procaine block of the sympathetics is worth while Whenever possible, continuity of the carotid artery should be restored by lateral suture endarterectomy or by interposition of a substitute segment.

* The diagrams and legends in this chapter have been constructed by the author for the Subcommittee on Vascular Injuries of the Committee on Surgery, Division of Medical Science of the National Research Council They were published in the Military Surgical Manuals of the National Research Council, volume 5, W B Saunders Company, Philadelphia and London, 1943 Some new diagrams have been added here These diagrams not only facilitate exposure but help in the interpretation of arteriograms

Figure 331. Exposure of the Subclavian Artery

A. Recline the patient's head and turn it to the opposite side. Place sandbags under his shoulder blades

B. Incise parallel to, and one fingerwidth above the clavicle over the clavicular insertion of the sternocleidomastoid muscle to the edge of the trapezius.

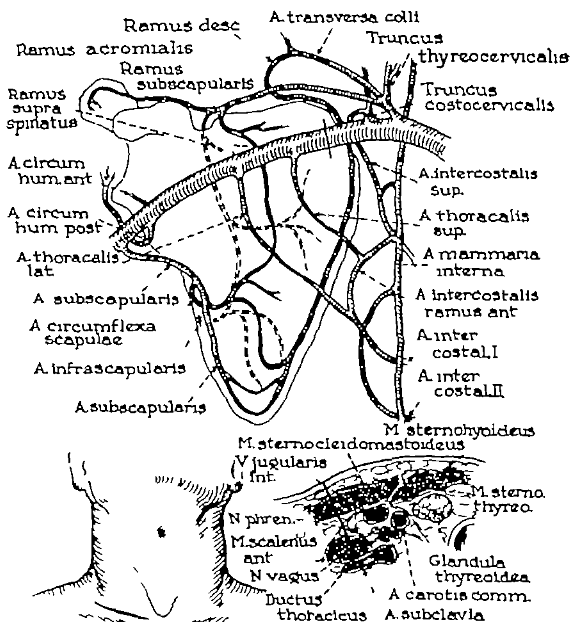
C Section the skin, platysma and branches of the supraclavicular nerves The external jugular vein need not be cut, it is better to ligate between ligatures, since nicking the vein may produce air embolism

D Section the sternocleidomastoid muscle to its sternal portion; retract the internal jugular vein medially.

E. Clear loose fat from the anterior scalenus muscle, define and retract the phrenic nerve medially.

F. Sever the anterior scalenus muscle with its posterior fascia The subclavian artery lies immediately behind it, over the apex of the pleura. The subclavian vein lies anterior to the scalenus muscle, between it and the clavicle The brachial plexus lies lateral to the artery, partially covered by the anterior scalenus muscle

UNFAVORABLE EFFECTS In case of arterial obstruction, the function of the arm is markedly impaired, although there is considerable collateral circulation In chronic occlusions, as in the aortic arch syndrome, intermittent claudication and Raynaud's syndrome develop



Ramus desc
A. transversa colli
Truncus
thyreocervicalis
Ramus acromialis
Ramus
subscapularis
Truncus
costocervicalis
Ramus
supra
spinatus
A. circum
hum. ant
A. intercostalis
sup.
A. circum
hum post
A. thoracalis
sup.
A. thoracalis
lat.
A. mammaria
interna
A. subscapularis
A. intercostalis
ramus ant
A. circumflexa
scapulae
A. inter
costal. I
A. infrascapularis
A. inter
costal. II
A. subscapularis
M. sternohyoideus
M. sternocleidomastoideus
V. jugularis
int.
M. sterno.
thyreo.
N. phren.
M. scalenus
ant
Glandula
thyreoidea
N. vagus
Ductus
thoracicus
A. carotis comm.
A. subclavia

Figure 331

Figure 332. Exposure of the Axillary Artery

A. Elevate the thorax and moderately abduct the arm.

B Incise the skin in the groove between the deltoid and major pectoral muscles, beginning at the clavicle, for a length of 8 to 10 cm. (3 to 4 inches).

C Retract the edges of the two muscles and identify the transverse fibers of the minor pectoral muscle

D Identify and retract the cephalic vein Visible in the loose fat above the free edge of the minor pectoral muscle are the brachial plexus, more medially the axillary artery, and medial to it the axillary vein.

UNFAVORABLE EFFECTS In case of axillary artery occlusion distal to the origin of the subscapular and circumflex branches, the viability of the upper extremity is in danger Certainly aspiration of clots, repair of associated nerve injuries and immediate dorsal sympathectomy or nerve block minimize the hazard

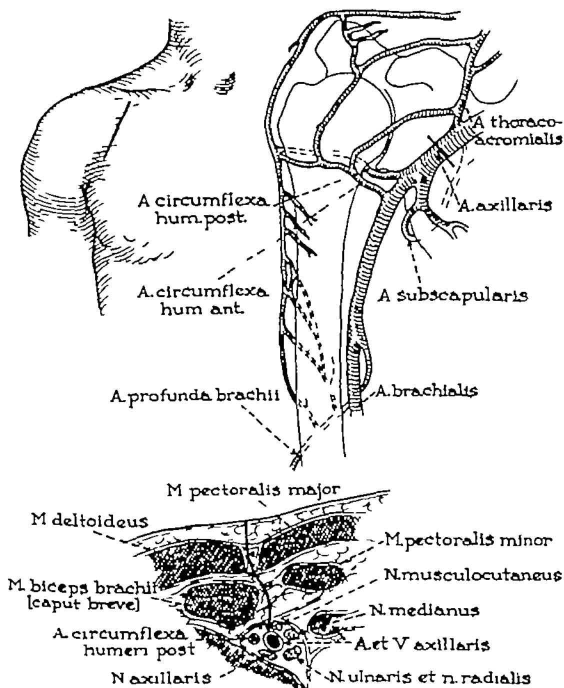

A thoraco-acromialis
A circumflexa hum. post.
A. axillaris
A. circumflexa hum ant.
A subscapularis
A. profunda brachii
A. brachialis
M pectoralis major
M deltoideus
M. pectoralis minor
N. musculocutaneus
M. biceps brachii [caput breve]
N. medianus
A. circumflexa humeri post
A. et V axillaris
N axillaris
N. ulnaris et n. radialis

Figure 332.

Figure 333. Exposure of the Brachial Artery

A. Abduct the arm in maximal supination

B Incise the skin in the middle of the upper arm over the bicipital groove

C Enter the fascia at the medial edge of the biceps and retract this muscle

D Isolate the thin, cutaneous antibrachii medialis nerve and the much heavier median nerve Retract the median nerve medially, the brachial vessels are in its close proximity High division into radial and ulnar arteries may occur

UNFAVORABLE EFFECTS Ligation below the profunda brachii and the superior ulnar collateral is fairly well tolerated, but restoration of continuity should always be attempted

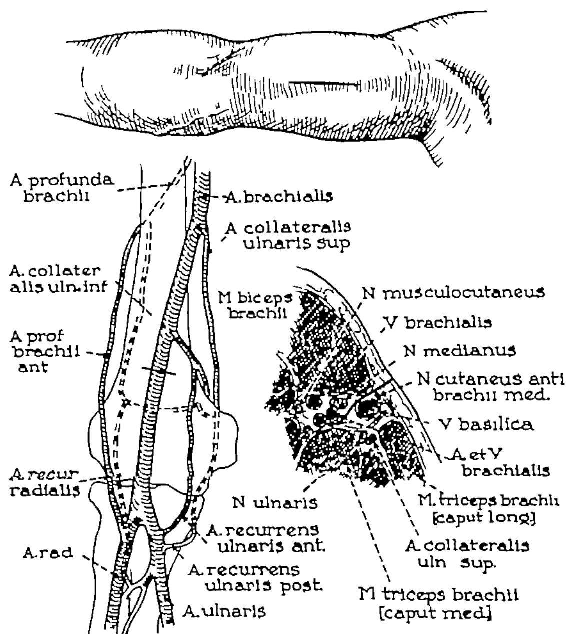

A profunda brachii
A. brachialis
A collateralis ulnaris sup
A. collateralis uln. inf
A prof brachii ant
A. recur radialis
A. rad
A. recurrens ulnaris ant.
A. recurrens ulnaris post.
A. ulnaris
M biceps brachii
N musculocutaneus
V brachialis
N medianus
N cutaneus anti brachii med.
V basilica
A. et V brachialis
N ulnaris
M. triceps brachii [caput long]
A. collateralis uln sup.
M triceps brachii [caput med]

Figure 333

Figure 334. Exposure of the Cubital Artery

A Abduct and supinate the arm

B Bisect the lacertus fibrosus through an incision running from the bicipital groove to the edge of the biceps tendon

C Ligate and cut the median cubital vein

D The median nerve is medial, lateral to it is the vein, and between and behind it is the artery If the incision is too low the pronator teres muscle is in the way.

UNFAVORABLE EFFECTS There is abundant collateral supply unless the recurrent vessels are destroyed below the bifurcation of the cubital artery.

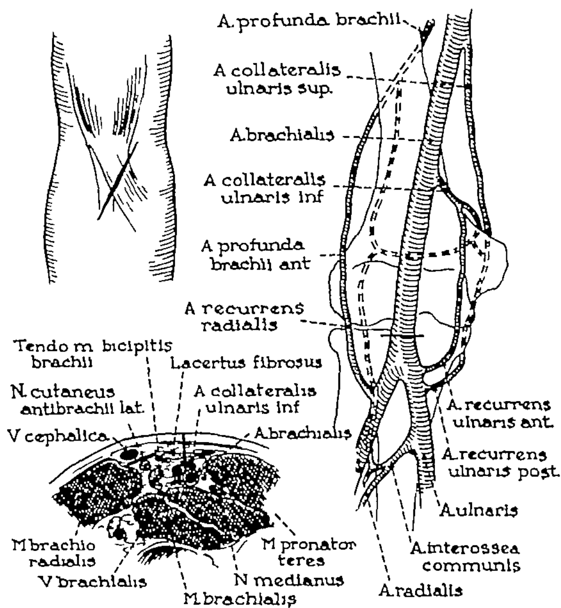

A. profunda brachii
A collateralis ulnaris sup.
A. brachialis
A collateralis ulnaris inf
A profunda brachii ant
A recurrens radialis
Tendo m bicipitis brachii
Lacertus fibrosus
N. cutaneus antibrachii lat.
A collateralis ulnaris inf
V cephalica
A. brachialis
A. recurrens ulnaris ant.
A. recurrens ulnaris post.
A. ulnaris
M brachio radialis
V brachialis
M brachialis
N medianus
M pronator teres
A. interossea communis
A. radialis

Figure 334

Figure 335. Exposure of the External Iliac Artery

A Place the patient flat on his back with a sandbag under his pelvis

B Incise the skin from the anterior iliac spine to the pubic tubercle, three fingerwidths above and parallel to the inguinal ligament.

C Tie and cut the superficial epigastric vessels and sever in the same direction the aponeurosis of the external oblique, cutting across the internal oblique and transversalis muscles and the transversalis fascia Do not open the peritoneum

D With the two index fingers bluntly dissect off the peritoneum from the psoas muscle and retract it medially and cephalad The ureter should be retracted with the peritoneum On the right, the vena cava divides just behind the common iliac artery The right common iliac vein first lies lateral to the artery, then passes behind it to its medial side The left common iliac vein lies altogether medial to the artery

UNFAVORABLE EFFECTS When two important collateral vessels, namely the deep circumflex and the deep epigastric, are open, through them the hypogastric artery can feed the femoral Occlusion of the external iliac artery above these vessels is better tolerated

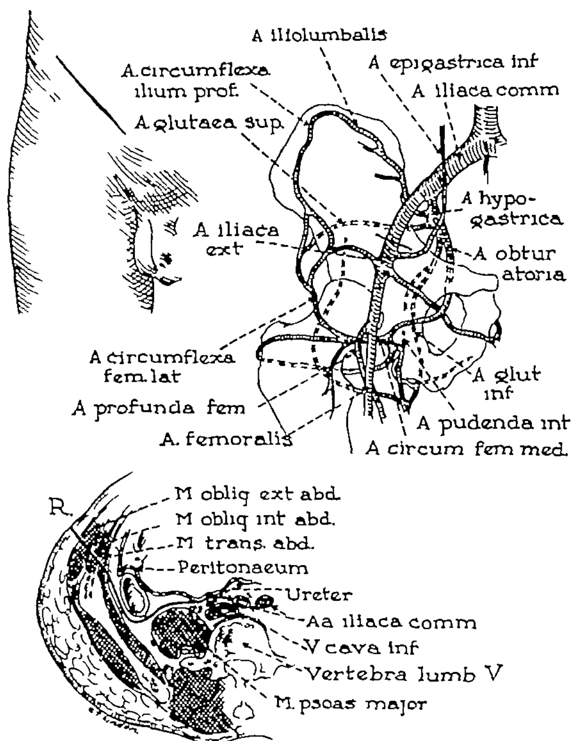

A iliolumbalis
A circumflexa ilium prof.
A epigastrica inf
A iliaca comm
A glutaea sup.
A hypogastrica
A iliaca ext
A obturatoria
A circumflexa fem. lat
A glut inf
A profunda fem
A pudenda int
A. femoralis
A circum fem med.
R.
M obliq ext abd.
M obliq int abd.
M trans. abd.
Peritonaeum
Ureter
Aa iliaca comm
V cava inf
Vertebra lumb V
M. psoas major

Figure 335.

Figure 336. Exposure of the Common Femoral Artery

A. Place the patient flat on his back with a sandbag or kidney rest to hyperextend his thigh

B Incise the skin from the middle of the inguinal ligament in a longitudinal direction for 10 cm (or, alternately, parallel to and two fingerwidths below the inguinal ligament).

C Identify the inguinal ligament and clear the fascia lata of fat and lymph nodes just below it

D Incise the fascia lata longitudinally The femoral vein is most medial, next lies the artery, and most lateral lies the nerve The major saphenous vein leads to the femoral

E Collateral circulation is established through the inferior gluteal and the medial circumflex to the popliteal artery Ligation below the profunda opens another important channel to the popliteal through a perforating branch. Tie, if possible, below the profunda. Suture the artery or insert a segment, if at all possible

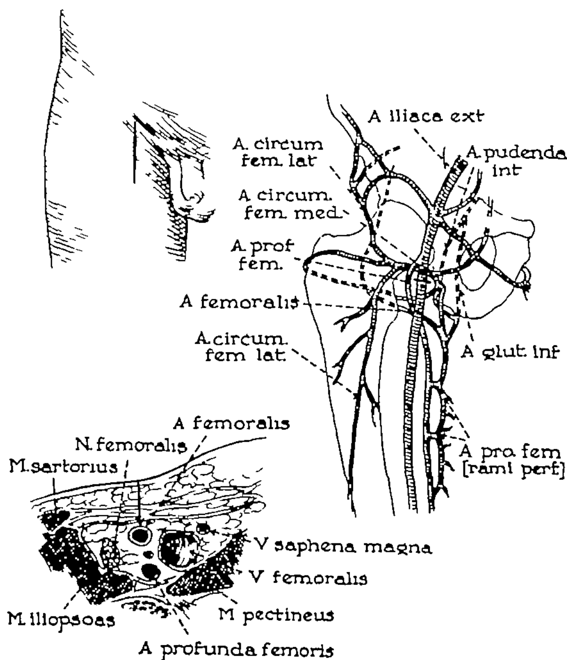

A iliaca ext
A. circum fem. lat
A. pudenda int
A. circum. fem. med.
A. prof fem.
A femoralis
A. circum. fem lat.
A glut. inf
A femoralis
N. femoralis
M. sartorius
A pro. fem [rami perf]
V saphena magna
V femoralis
M. iliopsoas
M pectineus
A profunda femoris

Figure 336.

Figure 337. Exposure of the Popliteal Artery

A Place the patient on his abdomen with a sandbag under his knee

B Make a generous longitudinal incision through the middle of the popliteal space, incising the fascia, the transverse incision is more popular Tie or retract the small saphenous vein

C In the loose fat closer to the lateral wall of the space (biceps), the tibial nerve is encountered, isolated with a nerve tape and laterally retracted Below and medial is the vein; deepest and most medial is the artery

D Collateral supply is poor at this level The channels operating on the closure of the femoral enter the popliteal artery above the fossa where there are no large masses of muscles and, therefore, the collaterals are small Ligature, if unavoidable, should be placed above the inferior collaterals Arterial suture is preferable to ligation Ligation is tolerated in aneurysms but the limb remains ischemic Grafting should always be attempted

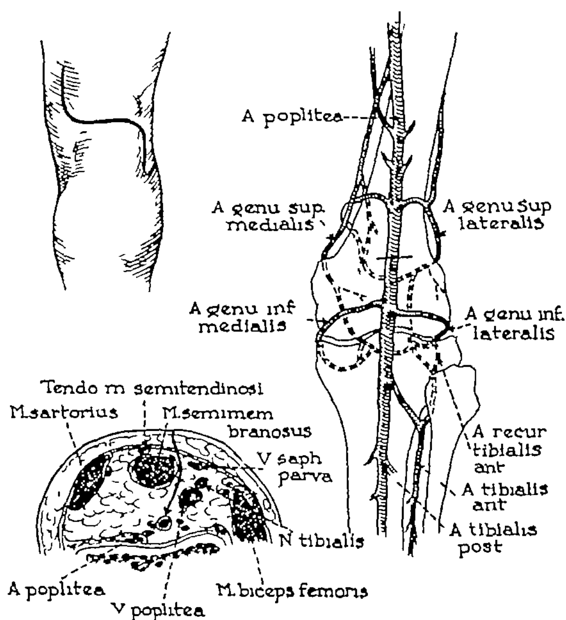

A poplitea
A genu sup medialis
A genu sup lateralis
A genu inf medialis
A genu inf lateralis
A recur tibialis ant
A tibialis ant
A tibialis post
Tendo m semitendinosi
M.sartorius
M.semimem branosus
V saph parva
N tibialis
A poplitea
V poplitea
M.biceps femoris

Figure 337

Figure 338. Exposure of the Anterior Tibial Artery

A Place the knee in full extension and the patient on his back

B Incise the skin in the middle of the lower leg one thumbwidth lateral to the lateral margin of the tibia Make the length of the incision 8 to 10 cm (3 to 4 inches)

C Split the muscle and fascia, and carefully identify the muscle space between the anterior tibial and the long extensor digitorum muscles.

D Retract these apart and expose within the cylinder of fat the vein, artery and nerve, which approach the tibia from above downward There is abundant collateral circulation from the posterior tibial and peroneal arteries Ligation of both anterior and posterior tibial arteries is safe only if the peroneal artery is intact

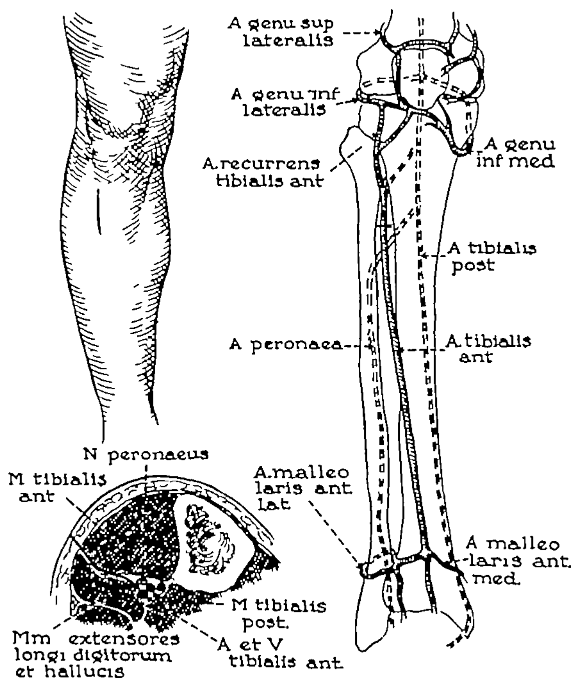

A genu sup
lateralis
A genu inf
lateralis
A.recurrens
tibialis ant
A genu
inf med
A tibialis
post
A peronaea
A tibialis
ant
N peronaeus
M tibialis
ant
A.malleo
laris ant
lat
A malleo
laris ant
med
M tibialis
post
Mm extensores
longi digitorum
et hallucis
A et V
tibialis ant

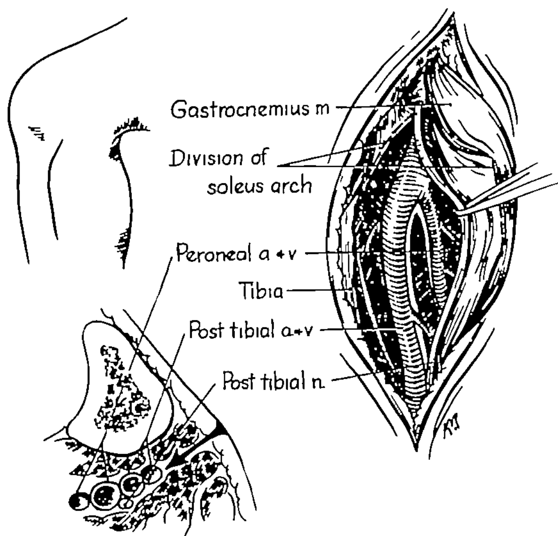
Figure 338

Figure 339. Exposure of the Tibioperoneal Trunk, Medial Approach

A Rotate the thigh externally and place a sandbag under the bent knee

B Incise the skin immediately below the level of the knee joint, two fingerwidths behind the inner edge of the tibia for about 3 to 4 inches (10 cm)

C Detach the thin longitudinal attachment of the soleus from the medial edge of the tibia, and continue cephalad, cutting the medial pier of the soleus arch In front of this lies the neurovascular bundle of the popliteal artery, vein and nerve and their branches Toward the tibia, the flexor digitorum longus and the posterior tibial muscles become visible. The neurovascular bundle lies against the posterior tibial muscle The tibial nerve should be carefully isolated. The peroneal vessels are found deeper in the wound The main collateral of the posterior tibial artery, outside the anterior tibial artery, is the peroneal branch When both posterior tibial and peroneal arteries are injured, the ischemia of the limb may become quite pronounced



Gastrocnemius m
Division of soleus arch
Peroneal a + v
Tibia
Post tibial a + v
Post tibial n

Figure 339

LIGATION AND STRIPPING OF THE SUPERFICIAL VENOUS SYSTEM

Figure 340. Ligation of the Long Saphenous Vein at the Groin

A. Make a hockey stick incision starting three fingerwidths above the inguinal ligament and curving medially below the ligament The incision is placed one fingerwidth medially to the palpable femoral artery. In thin individuals, an incision parallel to and just below the inguinal ligament is acceptable

B. The terminal portion of the long saphenous vein is exposed, visualizing the variable pattern of tributaries and transecting them between no 100 cotton ligatures The external pudendal artery is isolated and, should it lie anteriorly to the saphenofemoral junction, it too is cut The femoral vein must be well visualized both above and below the junction

C The saphenofemoral junction is free and ready to be tied An artery clamp is placed on its distal end.

D. The vein has been ligated with no 40 cotton flush with the femoral vein. A generous (½ inch) stump is left, to be transfixed with a stick tie The distal end awaits the arrival of the stripper from the ankle

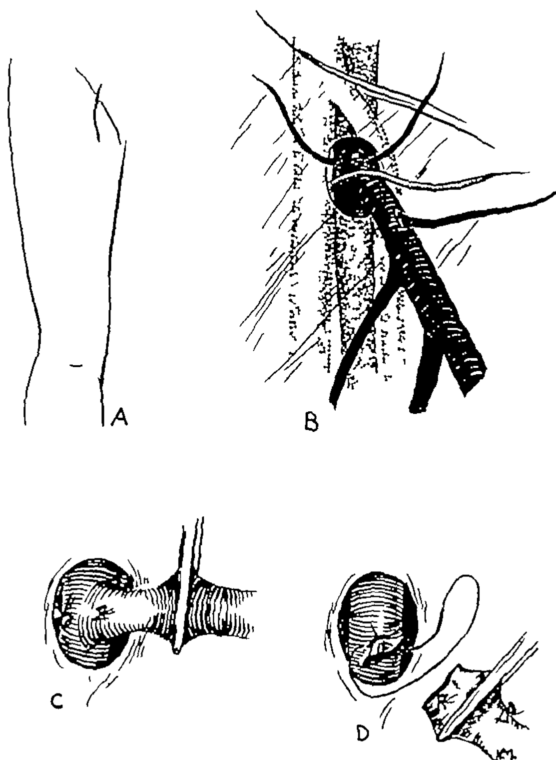


Figure 340

Figure 341. Stripping of the Long Saphenous Vein

A. While one surgeon exposes the saphenofemoral junction in the groin, another makes a small longitudinal incision below the inner malleolus, on the *dorsum of the foot* and not at or above the level of the ankle.

B. All tributaries having been tied, a no 60 cotton ligature is placed on the distal end of the long saphenous vein A small transverse nick is made in the vein with a "plastic" scissors and the flexible Myers stripper is advanced toward the groin Should it meet obstruction, which is either a kink, an aneurysmal dilatation or a thrombus, a small longitudinal incision exposes its head and the stripper is extracted from below upward, after the olive at the ankle is tightly secured around the vein with three no 8 cotton ligatures

C The head of the stripper has arrived to the groin and is promptly extracted In case an inadequate saphenous ligation has been done before, the insertion of the stripper and the sounding of the vein is the first step in the operative procedure

D. The patient is placed in a Trendelenburg position and the vein is extracted from below upward Manual compression with sterile towels is exerted for five minutes to minimize the formation of a hematoma in the bed of the extracted vein All skin incisions are closed with interrupted mattress sutures. Subcutaneous stitches are only used in the groin. The skin around the incision is painted with Mastisol,* and elastoplast strips are applied for dressing Large laparotomy pads are placed around the entire extremity and 4-inch wide elastic bandages from toes to groin complete the dressing

E The stripped vein on the stripper is examined for tears and tributaries It is not saved for arterial grafting as in the past, since it has proved unsuitable

* Gum mastic 40 Gms, benzol 60 Gms, castor oil, 20 drops This keeps indefinitely at room temperature, and is painted on the skin with two applicators

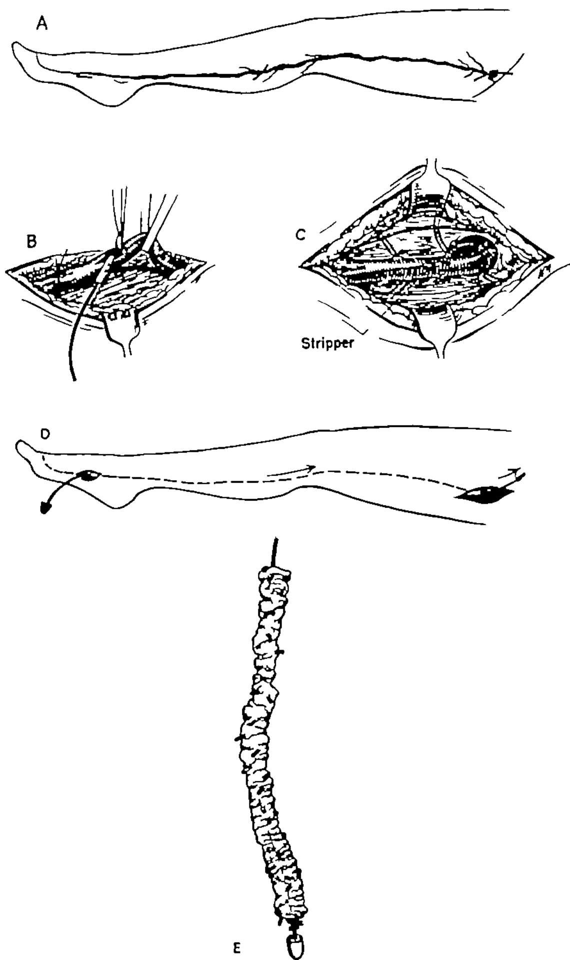


Figure 341

Figure 342. Ligation and Stripping of Short Saphenous Vein

A. A small longitudinal incision is made below the level of the external malleolus, between it and the Achilles tendon.

B The skin incision is deepened and exposes the short saphenous vein which is often tortuous and which shows many tributaries

C The short saphenous (sural) nerve is carefully kept out of the field, since it is easily traumatized or cut Its injury leads to long-lasting if not permanent anesthesia or paresthesia A flexible stripper with a small olive is then inserted proximally into the incised saphenous vein It passes cephalad and may stop below or well above the knee level, depending on the pattern of the short saphenous vein The skin is incised longitudinally above or below the popliteal fossa, and transversely at the popliteal level, the stripper is extracted and the vein pulled out with it. The proximal end of the vein is ligated as close to its entrance into the deep vein as possible. This may not always be feasible at midthigh, unless long incisions are made

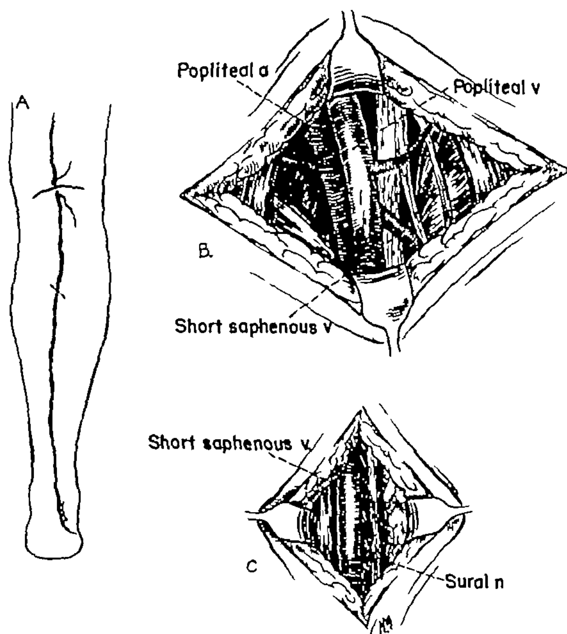

A
Popliteal a
Popliteal v
B
Short saphenous v
Short saphenous v
C
Sural n

Figure 342.

Figure 343. Ligation of Incompetent Perforators

A The patient's skin is marked with crosses at the level of suspected incompetent perforators * Longitudinal incisions are made at the level of a palpable defect in the fascia If there are several incompetent perforators with a consecutive mass of varicosities, one long incision is made.

B The "hole" in the fascia is palpated and the tortuous bulbous vein is dissected down to the fascia, usually there is a dilated segment dividing into several branches

C The incompetent perforator vein is tied flush with the fascia, and the spongy mass of varicosities is excised.

D Closure is made with interrupted mattress sutures.

E If there is a great number of perforators, a long *subfascial* incision is made, either on the medial side of the leg from the malleous to a level close to the knee, or through a stocking seam incision on the posterior surface of the calf (fig 344)

* For an indelible dye, a solution of pyrogallic acid 5 Gm , acetone 50 Gm., sol of ferric chloride 40 Gm and ethyl alcohol to make 100 cc is used (T T Myers, M D)

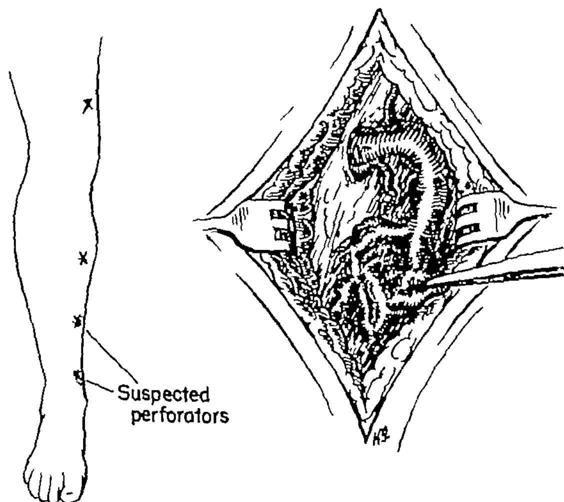


Figure 343

Figure 344. Subfascial Ligation of All Lower Leg Perforators

A A longitudinal incision is made from the popliteal fossa to the ankle level in the midline The incision is deepened through the deep fascia, and the short saphenous vein and the sural nerve are exposed.

B The short saphenous vein is excised completely, carefully protecting the sural nerve

C Subfascially, the medial lateral and the anterior groups of perforators are doubly ligated and cut If the fascia is thickened, a wide strip of it is excised at the same time

D. Subcutaneous and cutaneous stitches close this "stocking seam" incision Snug dressing is applied

E In a cross section, the communications of the superficial veins with the anterior tibial, peroneal and posterior tibial veins are illustrated. (Felder, D A , Murphy, T. O. and Ring, D M Posterior Subfascial Approach to Communicating Veins of the Leg Surg Gynec. and Obst , *100* 730, 1955)

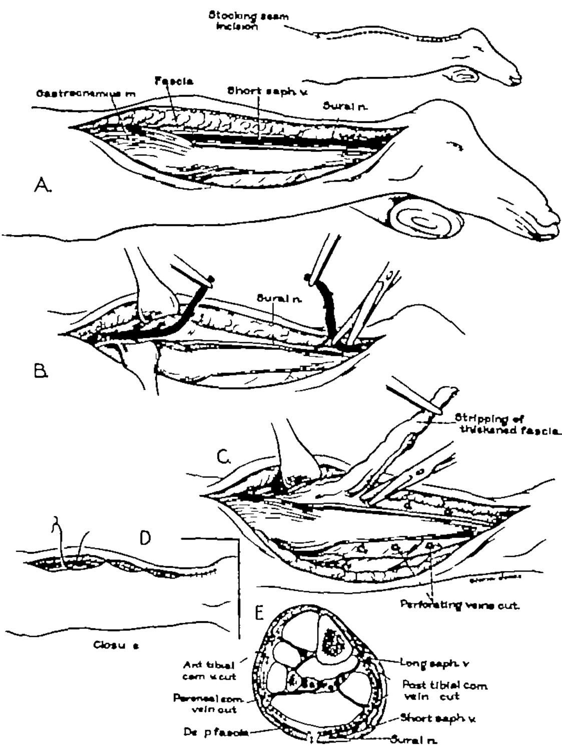

Stocking seam
incision
Fascia
Gastrocnemius m
Short saph. v.
Sural n.
A.
B.
Sural n.
C.
Stripping of
thickened fascia
D
Closu e
Perforating veins cut.
E
Art tibial
com v cut
Peroneal com.
vein cut
De p fascia
Long saph. v
Post tibial com
vein cut
Short saph. v
Sural n.

Figure 344

LIGATION OF DEEP VEINS

Figure 345. Ligation of the Vena Cava

A Place the patient on his back with a sandbag under the costal margin, the table moderately broken and the kidney rest elevated

B. A muscle-splitting extraperitoneal exposure on the right is done just as in a right lumbar sympathectomy

C With the help of a Mixter forceps, two no 20 cotton ligatures are placed between the lumbar veins and well below the entrance of the renal veins The vein need not be sectioned Should there be a clot at this level, this is first aspirated No attempt is made to preserve the continuity of the vena cava A distal thrombus may later produce pulmonary embolism

D A cross section shows the line of cleavage and the level of the ligature at L_2.

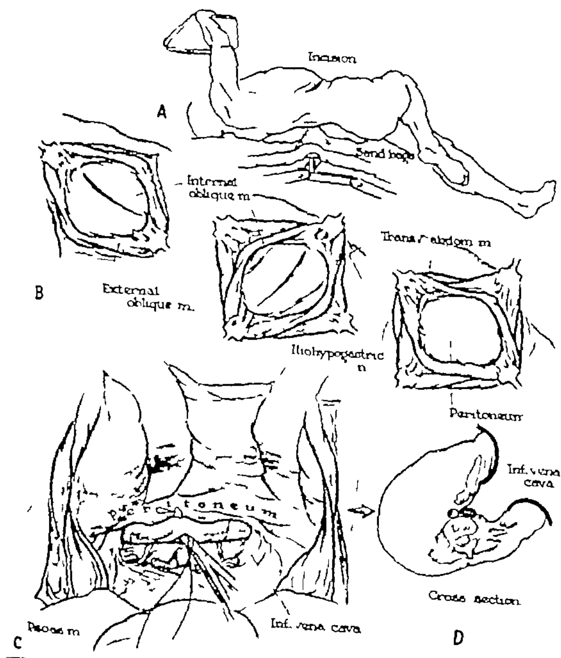

Incision
A
Sand bags
Internal oblique m
B
External oblique m.
Trans. abdom m
Iliohypogastric n
Peritoneum
Inf. vena cava
peritoneum
Cross section
Psoas m
Inf. vena cava
C
D

Figure 345

LIGATION OF DEEP VEINS

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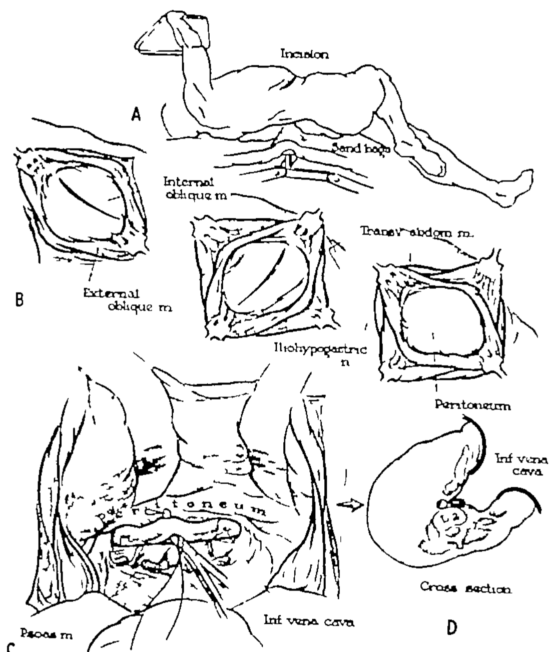

Incision
A
Sand bags
Internal oblique m
Transv abdom m.
B
External oblique m
Iliohypogastric n
Peritoneum
peritoneum
Inf vena cava
Cross section
Inf vena cava
Psoas m
C
D

Figure 345

Figure 346. Ligation of the Common Femoral Vein

A A longitudinal incision is made one-third above and two-thirds below the inguinal fold, along the course of the femoral artery

B The long saphenous vein is exposed, followed to its junction with the femoral vein, and liberally exposed above and below the junction.

C The vein is separated from the femoral artery, which is retracted with cord tape Note the saphenous nerve within the vascular sheath The surgeon ligates and cuts between the ligatures and above the deep femoral and long saphenous veins to avoid embolism from these sources. Subcutaneous sutures are placed to eliminate dead space and the skin is closed with interrupted cotton sutures

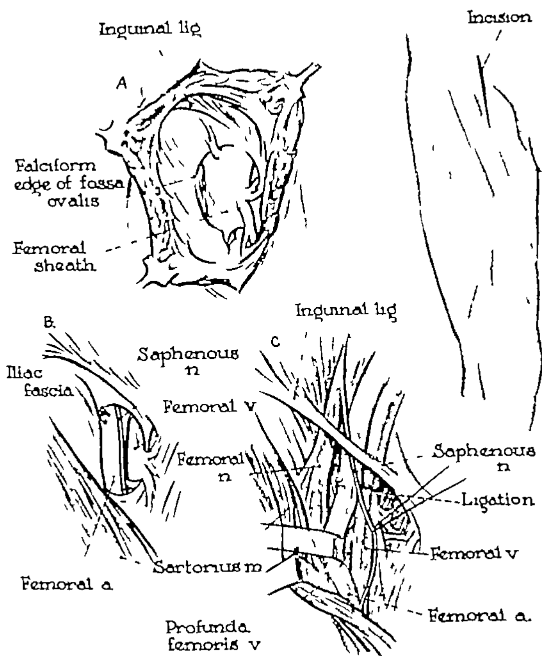

Inguinal lig
A
Falciform edge of fossa ovalis
Femoral sheath
Incision
B
Iliac fascia
Saphenous n
Femoral v
Femoral n
Sartorius m
Femoral a
Profunda femoris v
Inguinal lig
C
Saphenous n
Ligation
Femoral v
Femoral a.

Figure 346

Figure 347. Ligation of the Popliteal Vein

A. A longitudinal or transverse incision of the skin is made in the popliteal fossa.

B A longitudinal incision is made parallel to the short saphenous vein and nerve in the deep fascia

C The popliteal nerve is retracted and one or two popliteal veins are sectioned There may be several large collaterals when the vein has been occluded The artery lies deep and medial to the popliteal vein.

D. The deep fascia and skin are resutured

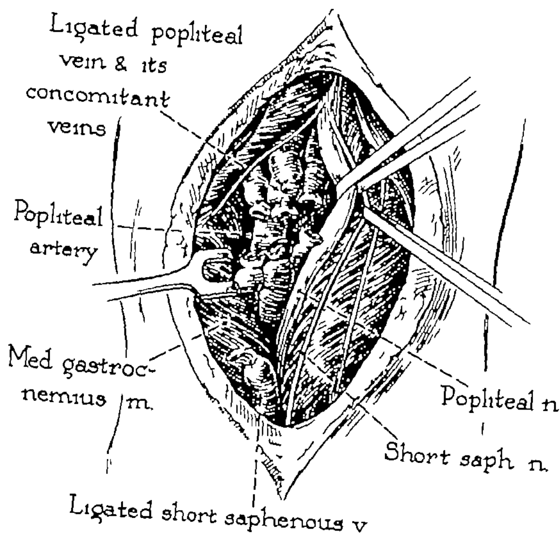

Ligated popliteal vein & its concomitant veins
Popliteal artery
Med gastrocnemius m.
Popliteal n
Short saph n.
Ligated short saphenous v

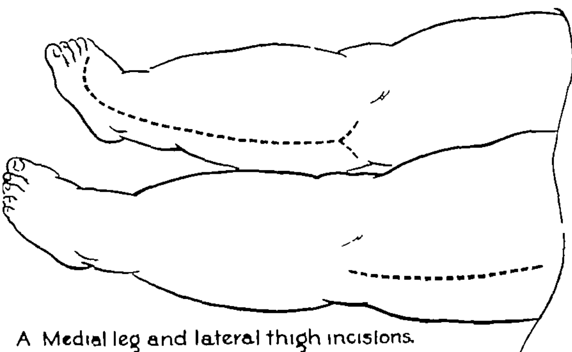
Figure 347

LYMPHANGIOPLASTY

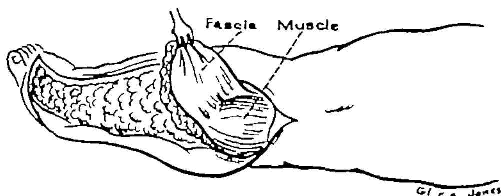
Figure 349. Lymphangioplasty with Preservation of Skin

A A medial incision in one calf may be combined with a lateral incision of the other calf or thigh done at the same time Thigh incisions have been more or less abandoned Note the incision on the dorsum of the foot

B Indurated fat, fibrous tissue and large lymph spaces are excised together with the deep fascia. The skin is then placed on the muscle, with multiple stab wounds to facilitate drainage (Homans, J Treatment of Elephantiasis of the Legs, Preliminary Report New England J Med. *215* 1099, 1936)



A Medial leg and lateral thigh incisions.



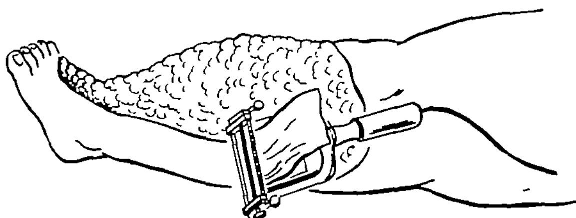
B Excision of superficial and deep fascia baring muscle.

Figure 349

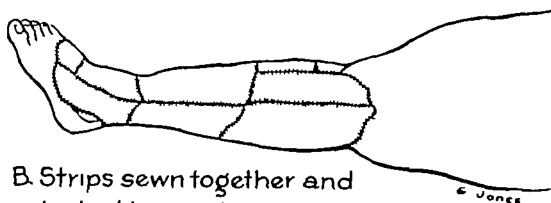
Figure 350. Lymphangioplasty with Removal of Split Thickness Skin

A. An electric dermatome removes the skin.

B The strips are sewn together and placed on the muscle which is denuded of fascia (Pratt, G H Surgical Correction of Lymphedema. J.A M A , *151* 888, 1953)



A Electric dermatome removes skin.



B. Strips sewn together and tacked to muscle

Figure 350

Figure 351. Lymphangioplasty with Replacement of Full Thickness Skin

A The skin is marked with indelible dye A circular incision is made at the level of the tibial tuberosity, descending vertically in the posterior midline to a point just below the malleoli, then swinging anteriorly just below the malleoli where it meets at the level of the toe webs

B. The skin, lymphedematous tissue and fascia are removed in one piece, starting at the knee where the fat is coned down to the level of the deep fascia The saphenous veins and superficial nerve trunks are sacrificed All tissue behind and above the malleoli is removed. On the dorsum of the foot and ankle, the paratenon should be left intact over the extensor tendons.

C Strips of fat are cleared with a knife or curved scissors down to the level of the dermis. All fatty remnants need to be carefully removed

D. The skin is now replaced and the excess trimmed away until a snug fit is obtained. Too little or too much tension must be avoided Stab wounds are placed with a no. 11 knife for drainage.

E The skin is carefully resutured, but resutured loosely at the posterior incision to allow for drainage. The skin is treated as a full thickness free graft with fine mesh gauze, much padding and snug but not tight elastic compression No posterior molded splint is applied (By Barron, J. N In Martin, Lynn, Dible and Aird, editors· Peripheral Vascular Disorders E S Livingston, Ltd , Edinburgh and London, 1956, p. 817)

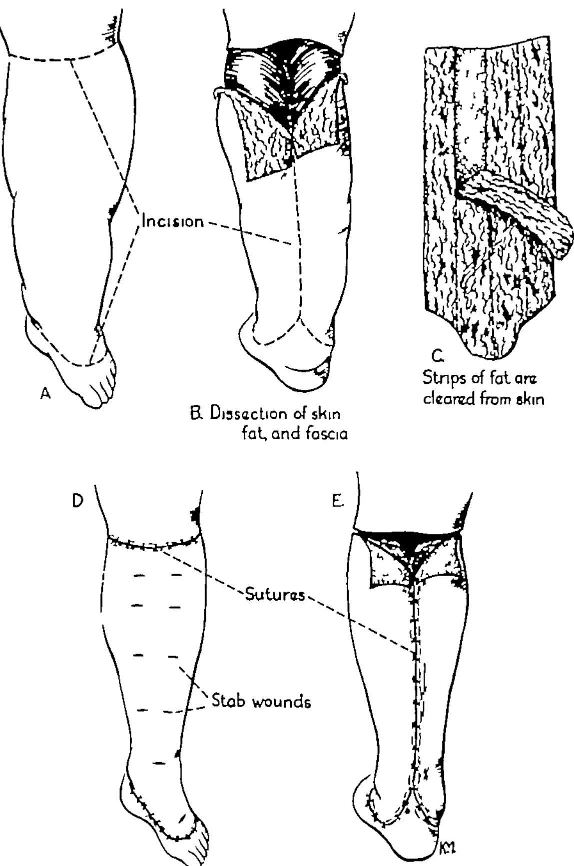

Incision
A
B. Dissection of skin fat, and fascia
C.
Strips of fat are cleared from skin
D
E
Sutures
Stab wounds
KM

Figure 351

Figure 352. Lymphatico-Venous Anastomosis

A large lymph channel in the left retroperitoneal space is anastomosed side to side to the hemiazygos vein to overcome obstruction at or below the cisterna chyli This may be used in cases of chylous reflux to the lower extremity, when ligation of lymph trunks in the groin does not decrease edema (Brewer, C A , III Surgical Management of Lesions of the Thoracic Duct Am J Surg , *90* 210, 1955)

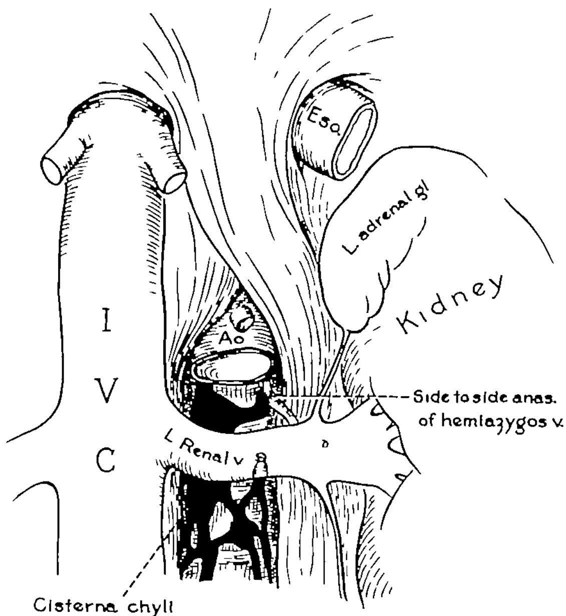

Eso
L adrenal gl
Kidney
I
V
C
Ao
Side to side anas.
of hemiazygos v.
L Renal v
Cisterna chyli
Gloria Jones

Figure 352.

ARTERECTOMY

Figure 353. Arterectomy

A An incision is made along the bicipital sulcus

B Then gently retract the median nerve

C Make a double ligation and excision of the brachial artery for injury, thrombosis or arterial disease

D This is always followed by restoration of continuity by end to end or end to side placement of a vascular graft or prosthesis Arterectomy alone has no detectable effect on improving circulation.

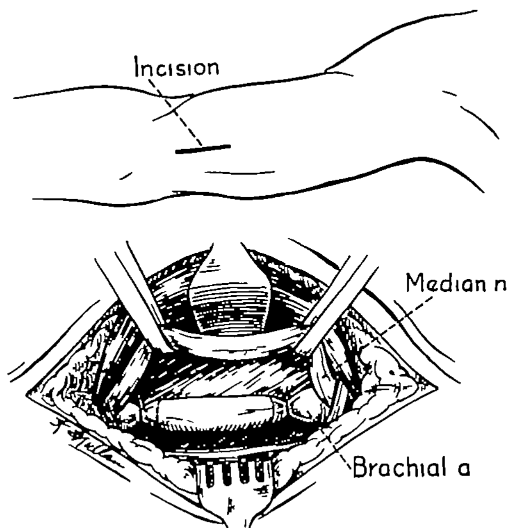


Figure 353.

ARTERIAL EMBOLECTOMY

Figure 354. Femoral Embolectomy

Embolectomy may be performed on any major artery, whose occlusion results in ischemia

A A longitudinal incision is made along the course of the femoral artery This may be extended cephalad to allow for retroperitoneal dissection of the iliac artery

B The common femoral artery is exposed The embolus is often at the bifurcation. The vessel is distended, soft and nonpulsatile.

C. Apply cord tapes or rubber catheters to the proximal segment and to the profunda femoris Incise the common femoral artery longitudinally and squeeze gently on the distal clot Should free bleeding occur, control it with a tape on the superficial femoral artery

D A proximal clot may have to be massaged down into the wound by gently inserting the second and third fingers retroperitoneally as high as possible

E If the clot is short, suction with a glass tip will deliver it Free, pulsatile flow of blood must appear, which is then arrested with tape. Bulldog clamps are convenient but they may cause damage, especially to sclerotic vessels Distal suction is most important, and if no free flow of blood appears, the popliteal or the posterior tibial artery is opened and the clot delivered by retrograde flush with a 50 cc syringe armed with a cannula

F Closure is made with an interrupted or continuous 00000 Deknatel silk suture or 00000 Mersilene (Dacron), swaged on a straight needle and carried without a needle holder

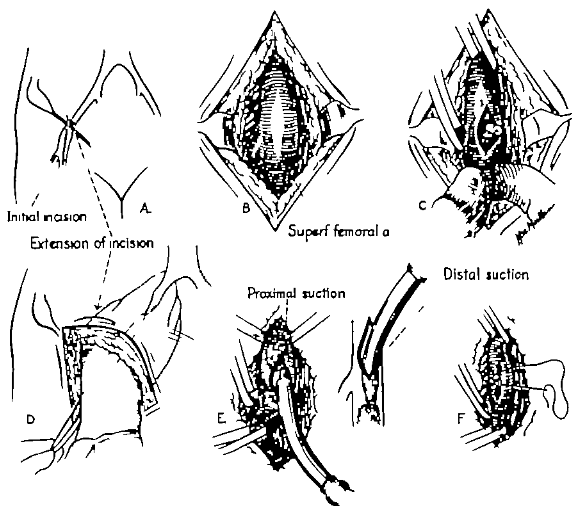

Initial incision
A
B
C
Superf femoral a
Extension of incision
Distal suction
Proximal suction
D
E
F

Figure 354

PULMONARY EMBOLECTOMY

Figure 355. Pulmonary Embolectomy

A. A submammary skin incision is made, no anesthesia is needed

B Open the chest between the fourth and fifth ribs, expose the pericardium and incise the anterior wall of the right heart longitudinally after placing two parallel silk sutures in its wall

C Push a no. 34 catheter or a metal tube through the cardiac incision and cross the sutures over the catheter to stop bleeding Milk the palpable clot in the pulmonary artery gently back into the ventricle and extract it by suction.

D. Close the incision in the right ventricle with some additional sutures and leave the pericardium slightly open for drainage

E Close the thoracic wall under water seal drainage. (Neuhof, H.: Problem of Embolism of the Pulmonary Artery. Ann Surg., *142* 568, 1955)

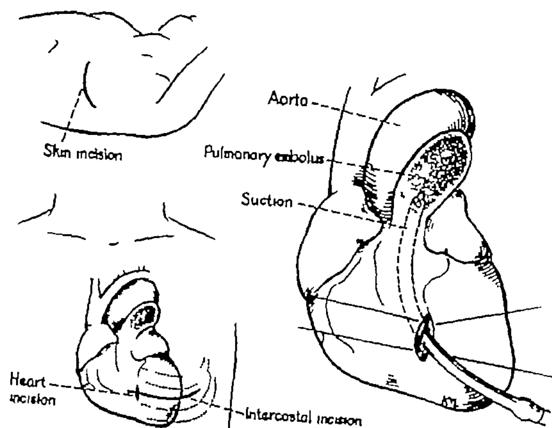

Skin incision
Aorta
Pulmonary embolus
Suction
Heart incision
Intercostal incision

Figure 355

Figure 356. Principles of Restoring the Blood Flow in Short Segmental Occlusions (Iliac or Femoral)

In a short segmental occlusion, the occluding atheroma or thrombus can be.

A. Reamed out

B Excised with direct anastomosis between the segments or with interposition of a graft, and

C Bypassed with an end to side technique The last procedure allows the use of wide prostheses and larger anastomoses, and is thus the best safeguard against late closure due to stricture at the anastomoses

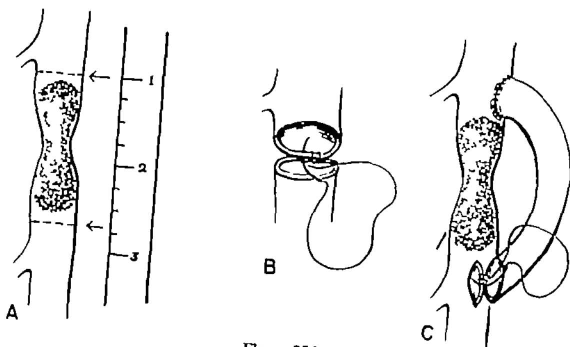


Figure 356

Figure 357. Femoral Thromboendarterectomy

A. Expose a normal or fairly normal area of the femoropopliteal segment. This may be done at the lower end of Hunter's canal or below the knee with an incision one thumbwidth medial to the tibial edge. Transect (do not incise) the occluded segment at its most distal portion and establish a line of cleavage with an arterial stripper

B Define and free the distal segment Suture the separated intima to the wall of the vessel to prevent dissection after the arterial flow has been established Incise the occluded femoral artery at a point where the stripper is stuck, or after the pulsatile flow has been established, usually at the common femoral level Extract the inner core in one or several segments and release the proximal clamp several times to flush out the artery from clots or granulation tissue

C Establish an end to end anastomosis between the reamed portion of the vessel and the distal segment, whose intima is securely in place and from which adequate backflow can be demonstrated Close the longitudinal incision in the proximal segment over the catheter Interrupted sutures are less apt to constrict the lumen than a continuous one.

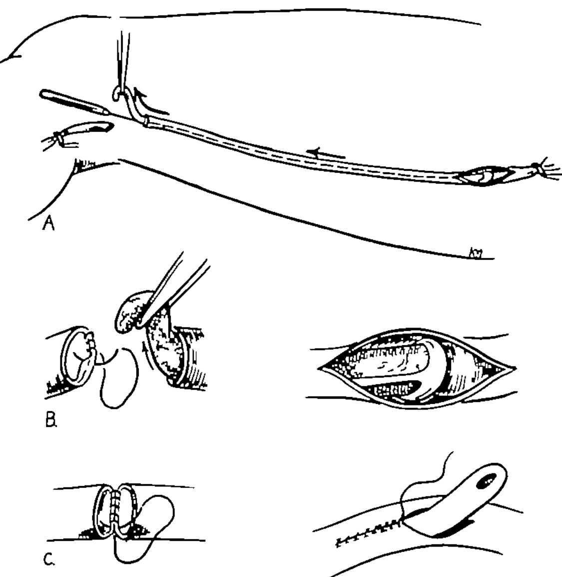


Figure 357

BYPASS PROCEDURES

Figure 358. End to Side Anastomosis in a Bypass Procedure

A A longitudinal incision is made in the host artery and is isolated by clamps or cord tape A small oval segment may be excised

B A longitudinal slit is made in the homograft, if a plastic prosthesis is used this is cut obliquely and the ends seared with a cautery to prevent fraying The corners of the arterial flap are trimmed

C Mattress sutures are placed at the two ends of the segment to be implanted

D A continuous over and over stitch unites the two mattress sutures

E The end to side anastomosis completed This is a wide anastomosis, the longitudinal incision being two to three times as long as the diameter of the vessel (Linton, R R. and Menendez, C V.. Arterial Homografts Ann Surg , *142* 568, 1955)

Figure 359. Bypass of a Thrombotic Occlusion of the Abdominal Aorta

A. A midline laparotomy is made from the ensiform cartilage to the symphysis pubis The patient is in the Trendelenburg position and the small intestines are packed in a cellophane bag Moderate 88° hypothermia is useful, but is not necessary A long incision is made over the posterior peritoneum to the adventitia of the aorta Periaortic lymph glands are often in the way. An aortic clamp is applied just below the left renal vein The proximal aortic segment and the ostium of the renal artery may have to be reamed out to obtain a good end to end anastomosis Distal incisions are made in the common iliac, external iliac or even in the common femoral arteries, depending on the availability of a soft, patent distal segment

B A bifurcation graft, either a frozen arterial homograft or one of the new prostheses of Nylon, Dacron, or Teflon, is placed end to side into the distal stumps No attempt is made to excise the occluded aorta or to separate it from the vena cava If superficial femoral occlusion exists, this can be bridged from the graft to the popliteal artery with a straight plastic tube

Figure 358.

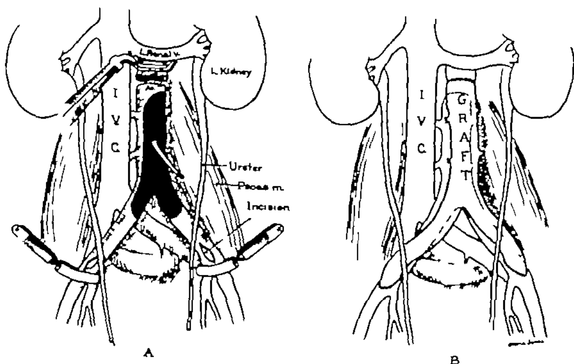


Figure 359

Figure 360. Resection of an Aneurysm of the Abdominal Aorta

A A midline laparotomy is made from the ensiform cartilage to the symphysis pubis Most aneurysms do not extend above the renal vein, but occasionally it becomes necessary to control the blood flow from above by direct pressure until the clamp can be satisfactorily placed. One or both of the common iliac arteries may be involved in the process, and the resection, of course, has to include them Often the aneurysm stops at the bifurcation. The part of the sac adherent to the vena cava is left attached to it. The hypogastrics need not be tied and cut unless they are closed If open, they are anastomosed to the graft

B The long bifurcation graft is implanted in place of the resected aneurysm. Distally, the anastomoses may be end to end or end to side. At this writing, crimped Dacron grafts are preferred, but the material is still subject to improvement

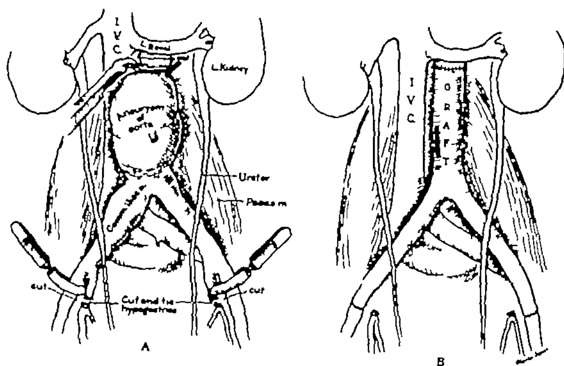


Figure 360

Figure 361. Resection of a Ruptured Abdominal Aneurysm

Massive transfusions are given to restore reasonable blood volume and renal function.

A Enter the left ninth intercostal space for digital compression of the thoracic aorta.

B A midline laparotomy is performed clamp the aorta below the renal vein and evacuate the retroperitoneal hematoma Then release thoracic compression

C When the bleeding is controlled, dissect the aneurysm and control both iliacs with tape or clamps

D Insert the crimped bifurcation graft In large aneurysms, the size of the proximal segment usually precludes the use of homografts (Modified from a diagram of Ethicon, Inc , Somerville, N J)

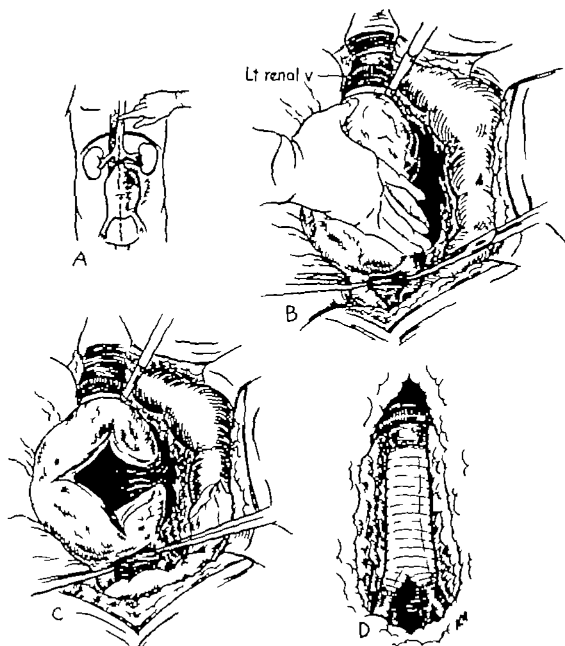


Figure 361

Figure 362. Obliteration of a False Lumen for a Dissecting Aneurysm of the Thoracic Aorta

A The relationship of the false and true lumens

B Clamps are applied above and below the transection of the aorta

C A cross section of the false and true lumens in the proximal and distal segments.

D A wedge is removed from the dissected inner wall of the proximal segment, *the false passage in distal segment is closed*

E End to end anastomosis is established for re-entry of the false passage into the true lumen (Reproduced by courtesy of Dr Oscar Creech, Jr , and the Texas State Journal of Medicine)

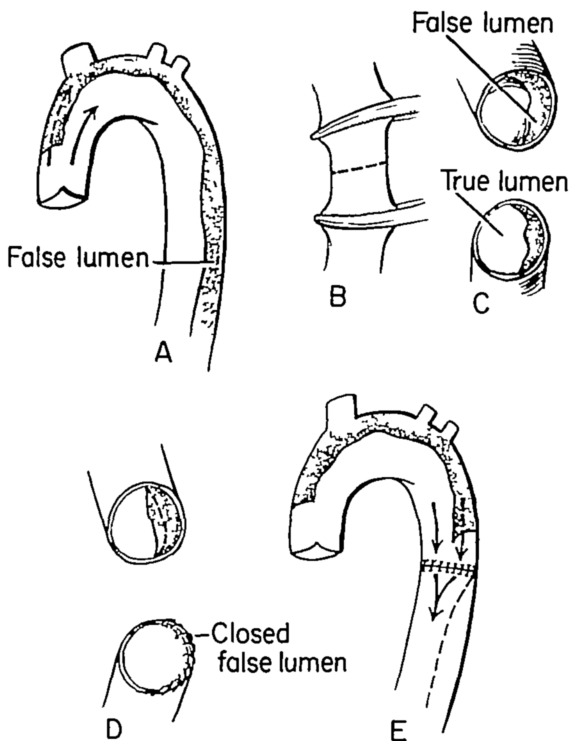

False lumen
True lumen
False lumen
A
B
C
Closed
false lumen
D
E

Figure 362.

e 362. Obliteration of a False Lumen for a Dissecting Aneurysm of the Thoracic Aorta

A The relationship of the false and true lumens

B Clamps are applied above and below the transection of the aorta

C A cross section of the false and true lumens in the proximal and l segments.

D A wedge is removed from the dissected inner wall of the proximal ent, the false passage in distal segment is closed.

E End to end anastomosis is established for re-entry of the false ige into the true lumen (Reproduced by courtesy of Dr. Oscar Creech, nd the Texas State Journal of Medicine)

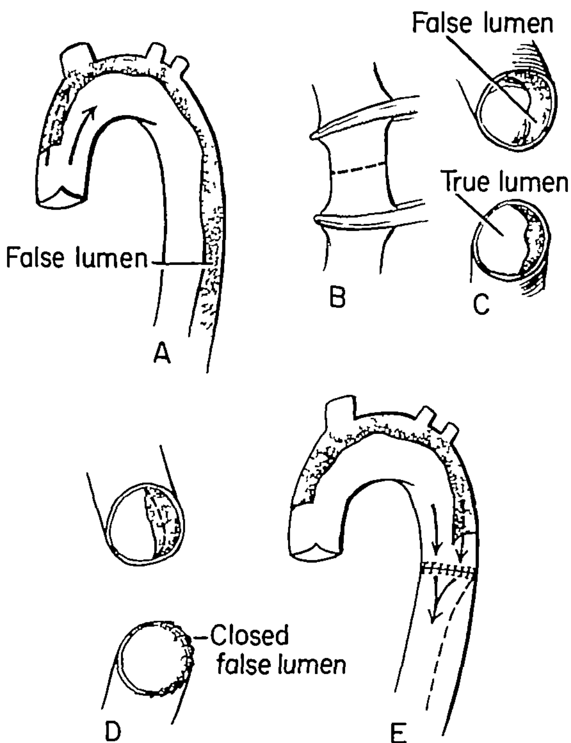

False lumen
True lumen
False lumen
A
B
C
Closed
false lumen
D
E

Figure 362.

Figure 363. Excision of Dissecting Aneurysms of the Aorta

A. The aneurysm starts distal to the origin of the left subclavian artery

B A single lumen is in the proximal segment, and a double lumen is in the distal segment The false passage in the distal segment will be obliterated

C Aortic continuity is restored by a homograft or by plastic material. (Modified from and reproduced by courtesy of Dr. Oscar Creech, Jr, and the Texas State Journal of Medicine)

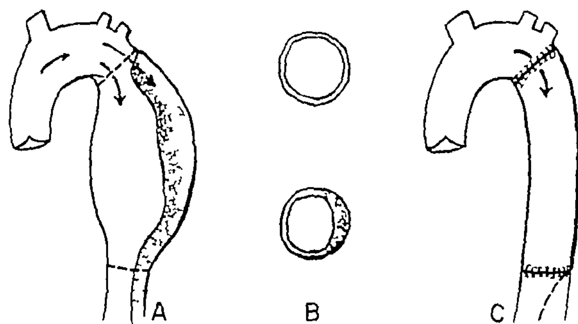


Figure 363

DENERVATION OF THE CAROTID SINUS

Figure 364. Denervation of the Carotid Sinus

A An incision is made at the level of the cricoid cartilage along the skin lines bisecting the pulsation of the carotid artery

B The platysma and the anterior sheath of the sternocleidomastoid muscle are cut and the muscle is bared The facial vein is seen at the medial edge of the wound

C. The vascular sheath is entered, exposing the common carotid artery at its bifurcation Note the important nerve structures to be saved.

D After infiltrating the area of bifurcation with 1 per cent procaine, which raises the patient's blood pressure, the triangular piece of tissue, including the carotid sinus nerves and the carotid body, is removed

E The adventitia is stripped for 1 inch proximally and distally from the bifurcation

F The extent of periarterial stripping is shown

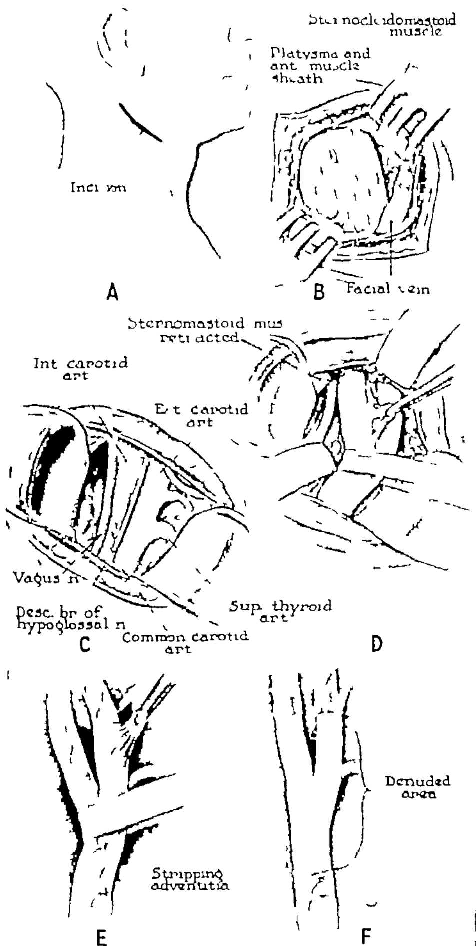

Sternocleidomastoid muscle
Platysma and ant muscle sheath
Incision
Facial vein
A
B
Sternomastoid mus retracted
Int carotid art
Ext carotid art
Vagus n
Desc. br of hypoglossal n
Common carotid art
Sup thyroid art
C
D
Denuded area
Stripping adventitia
E
F

Figure 364.

UPPER THORACIC SYMPATHECTOMY

Figure 365. Upper Thoracic Sympathectomy, Anterior Approach

A An incision is made one to two fingerwidths above the clavicle for a length of 8 cm. (a little more than 3 inches) Cervical and brachial plexus blocks with local infiltration can be used

B After cutting the skin and platysma, the clavicular portion of the sternocleidomastoid muscle is divided, as is the slender omohyoid

C The anterior surface of the scalenus anticus muscle is now cleared of fat and lymphatics, the phrenic nerve is retracted medially and the transverse cervical vessels are cut if they are in the way The scalenus muscle can now be cut, being careful not to injure the carotid sheath, which is retracted medially, and the subclavian artery which is below it

D Now the subclavian artery and the brachial plexus are in plain view The brachial plexus is severely left alone and not cleared It may be injected with 2 per cent procaine

E Before the subclavian artery can be retracted, Sibson's pleurocostal ligament has to be cut and the thyrocervical trunk divided After these maneuvers, the pleura is gently peeled back with the index finger or with a small wet sponge on a forceps (Hibbs' sponge).

F The apex of the pleura has been peeled away from the first rib and the costovertebral angle The deep wound can now be illuminated by a lighted retractor or head lamp The pleura can be dissected down below the third thoracic ganglion

G The chain is now cut below the third ganglion after a Cushing clip has been placed on it The rami entering the second and third ganglia are sectioned While originally the cut proximal end of the chain was implanted into muscle or into the intercostal nerve to produce preganglionic section, since 1940 the chain has been removed from below the third thoracic ganglion up to but not including the stellate and the intermediate ganglia If this is not seen, the vertebral artery is stripped and an extended cervicodorsal sympathectomy is done [3] (Gask with Telford's Modification Illustrated by de Takats, G Effect of Sympathectomy on Peripheral Vascular Disease Surgery, *2* *46*, 1937)

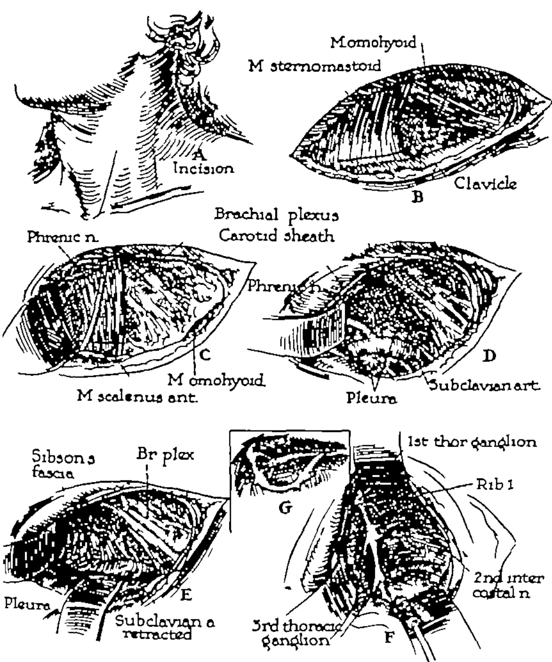

M omohyoid
M sternomastoid
A
Incision
Clavicle
B
Brachial plexus
Carotid sheath
Phrenic n
Phrenic n
C
D
M omohyoid
M scalenus ant
Pleura
Subclavian art
Sibson s fascia
Br plex
G
1st thor ganglion
Rib 1
2nd inter costal n
E
Pleura
Subclavian a retracted
3rd thoracic ganglion
F

Figure 365

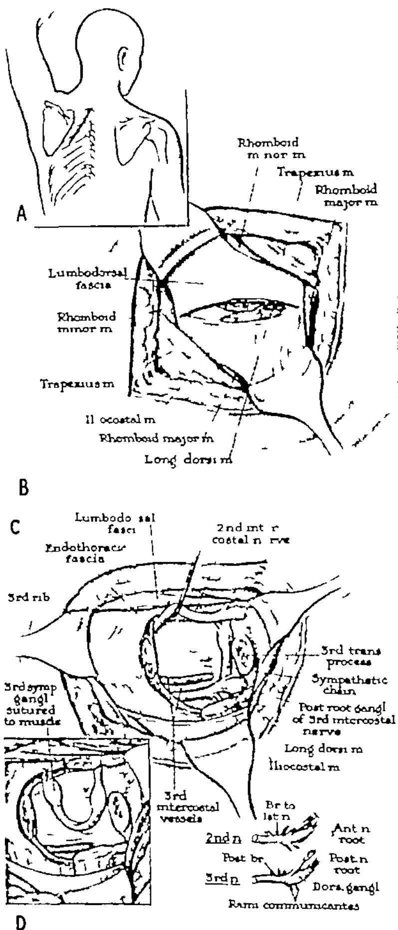
Figure 366. Upper Thoracic Sympathectomy, Posterior Approach

A The patient is in a prone position with an inflated horseshoe pillow under his chest His face is on a cerebellar rest The incision starts slightly below and lateral to the spinous process of the seventh cervical (prominens) vertebra and runs parallel to the medial border of the abducted scapula

B The trapezius muscle has been split in the direction of its fibers. The major and minor rhomboid muscles are retracted and the strong lumbodorsal fascia is split transversely The long muscles of the back are separated to expose the third rib.

C The third rib has been resected paravertebrally for a length of 6 cm The transverse process has been removed with a double action bone forceps The endothoracic fascia has been gently opened, which helps to peel off the pleura from the vertebral bodies The second and third intercostal nerves are pulled out gently from the intervertebral foramina so that the posterior root ganglion becomes visible With a small nerve hook the anterior and posterior roots can be separated, cut, and permitted to retract intradurally (see the insert at the lower right quadrant)

D The sympathetic chain is now cut below the third ganglion, well mobilized and implanted into the muscle As stated under the anterior approach (p 628), this preganglionic type of sympathectomy was abandoned since 1940, and the chain simply excised to, but not including, the stellate ganglion. The first thoracic ganglion, if it shows a slight indentation at its fusion with the inferior cervical ganglion, may be removed without the production of Horner s syndrome [4] (Smithwick's Method Illustrated by de Takats, G . Value of Sympathectomy in the Treatment of Buerger's Disease Surg Gynec and Obst , *79* 359, *1944*)



A
Rhomboid m nor m
Trapezius m
Rhomboid major m
Lumbodorsal fascia
Rhomboid minor m
Trapezius m
Il ocostal m
Rhomboid major m
Long dorsi m
B
C
Lumbodo sal fasci
2nd int r costal n rve
Endothoracic fascia
3rd rib
3rd trans process
Sympathetic chain
3rd symp gangl sutured to muscle
Post root gangl of 3rd intercostal nerve
Long dorsi m
Iliocostal m
3rd intercostal vessels
Br to 1st n
2nd n
Ant n root
Post br
Post n root
3rd n
Dors gangl
Rami communicantes
D

Figure 366.

Figure 367. Upper Thoracic Sympathectomy, Axillary Approach

A An incision is made in the axilla in the fourth intercostal space, extending slightly beyond the palpable margins of the trapezius and pectoralis muscles

B The two muscles are retracted, the fourth rib is defined and the long thoracic nerve is carefully isolated and protected.

C The pleura is entered in the fourth intercostal space and the ribs are gently but widely spread with a Finochietto ribspreader The parietal pleura is split over the thoracic sympathetic chain which is removed from below the azygos vein (fourth ganglion) to the slender neck of the stellate ganglion, seen at the top of the drawing The pleura is not resutured, and the thoracic wound is closed in layers. A mushroom catheter under water seal is left in for 48 hours (Atkins, H J B Sympathectomy by Axillary Approach Lancet, *1* 538, 1954)

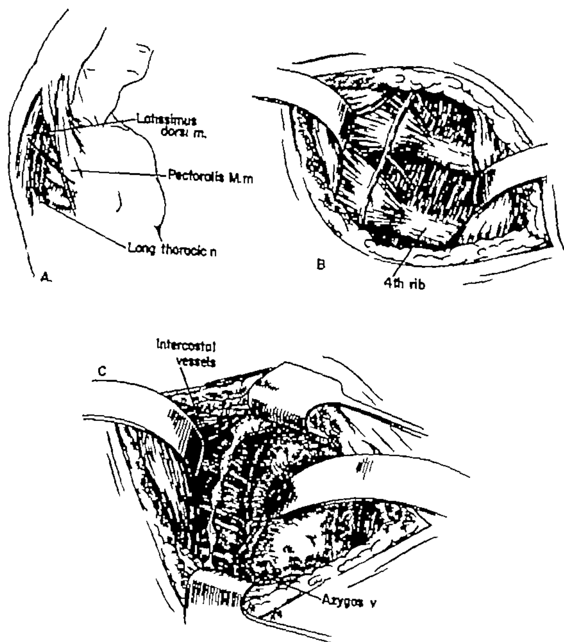

Latissimus dorsi m.
Pectoralis M.m
Long thoracic n
A
B
4th rib
Intercostal vessels
C
Azygos v

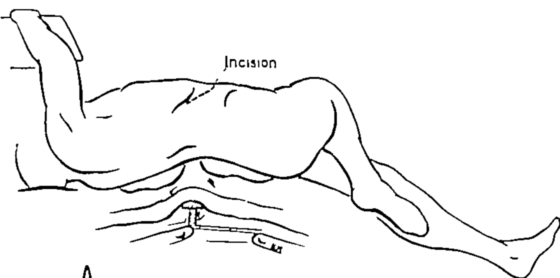
Figure 367

LUMBAR SYMPATHECTOMY

Figure 368. Lumbar Sympathectomy

A The patient's position on the table greatly facilitates exposure He is turned about 30° from the prone position, the table is moderately broken, the kidney rest is elevated at the level of the costal margin and there are sand-bags under his chest and buttocks His arm is on a flat table or trough. His hip and knee are flexed to 90° on the operated side, the opposite leg is straight

B. In such a position the psoas muscle is relaxed and the lumbar sympathetic chain is not covered by the tight fascial edge of the muscle.



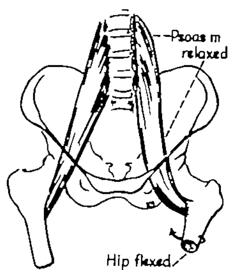


Figure 368.

Figure 369.

The skin incision, as shown in figure 368, extends from the midaxillary line bisecting the costal margin to the lateral edge of the rectus muscle

A The external oblique muscle and its aponeurosis are split along the line of the skin incision, with no undermining of skin edges

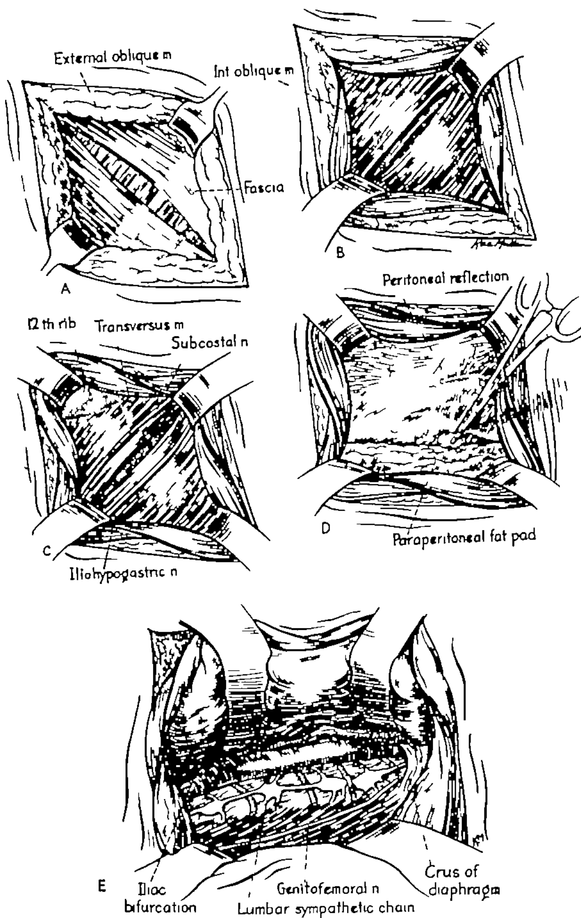
B After adequate blunt retraction, the internal oblique muscle comes into view Its thin, covering fascia is cut by a scalpel as close as possible to the tip of the twelfth rib and the muscle is separated bluntly by the two index fingers. Muscle relaxation must be complete, obtained either by spinal anesthesia to the level of the sixth dorsal segment or by inhalation anesthesia reinforced by curare

C After the internal oblique muscle is equally well retracted, an incision is made in the muscular and tendinous portions of the transversus muscle along the course of its fibers The incision must be parallel to but far enough from the subcostal and iliohypogastric nerves so that they suffer as little as possible from traction

D When this third muscle is retracted, the peritoneal sac comes into view This is gently peeled off with a small stick sponge toward the midline The dissection is started at a recognizable line of cleavage between the peritoneum and the paraperitoneal fat

E The peritoneal sac is now retracted with Deaver retractors toward the midline, cephalad to lift the kidney with its perirenal fat and caudad to expose the level of the aortic bifurcation On the right, the vena cava is retracted with a small stick sponge, on the left, the aorta does not cover the sympathetic chain The gangliononated trunk is now removed from the iliac bifurcation to the crus of the diaphragm The lumbar veins are usually under the chain, but frequently the second lumbar vein crosses *over* the chain. It is then doubly clipped with Cushing clips and cut

After the field is dry, the genitofemoral nerve and the cut ends of the trunk are flooded with 1 per cent procaine and the three abdominal muscles are united with interrupted no 40 cotton sutures If the subcutaneous fat layer is deep, it is brought together with a few interrupted no 60 cotton sutures The skin is closed with black cotton.



External oblique m
Int oblique m
Fascia
A
B
Peritoneal reflection
12 th rib
Transversus m
Subcostal n
C
Iliohypogastric n
D
Paraperitoneal fat pad
E
Iliac
bifurcation
Genitofemoral n
Lumbar sympathetic chain
Crus of
diaphragm

Figure 369

SPLANCHNICECTOMY AND DORSOLUMBAR SYMPATHECTOMY

Figure 370. Splanchnicectomy and Dorsolumbar Sympathectomy: Extent of Sympathetic Denervation for Hypertension

The diagram illustrates the various extents of sympathectomy Starting from the left, they are Peet's one-stage supradiaphragmatic splanchnicectomy; Adson's subdiaphragmatic approach, which has recently been revived with the addition of adrenalectomy, the transdiaphragmatic splanchnicectomy of Smithwick, Hinton's subtotal sympathectomy, extending from D_1 to and through L_3 and Grimson's total sympathectomy on the extreme right Of these operations, the transdiaphragmatic approach of Smithwick constitutes our routine procedure, if the patient has angina, postural hypertension or tachycardia, Hinton's transthoracic subtotal sympathectomy is used

Only the transdiaphragmatic approach will be illustrated The transthoracic approach has already been shown to the upper thoracic ganglia (de Takats, G Causes of Failure in Surgical Treatment of Hypertension Angiology, *1* 457, 1950)

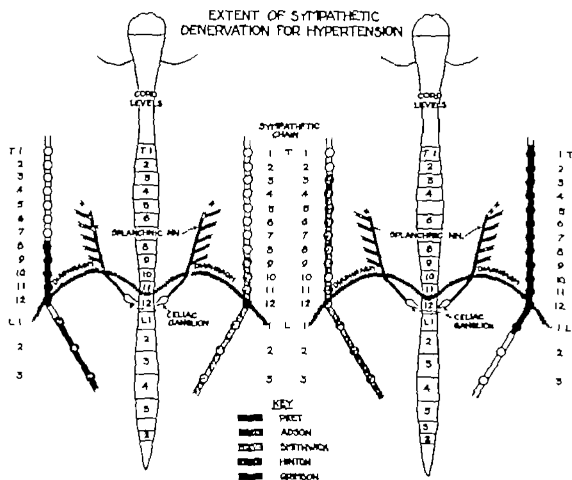

EXTENT OF SYMPATHETIC
DENERVATION FOR HYPERTENSION
CORD LEVELS
SYMPATHETIC CHAIN
SPLANCHNIC NN.
CELIAC GANGLION
KEY
PEET
ADSON
SMITHWICK
HINTON
GRIMSON

Figure 370

Figure 371. Transdiaphragmatic Splanchnicectomy and Dorsolumbar Sympathectomy

A. The patient is in a semilateral position, the table is broken and the kidney rest is elevated. If a bilateral one-stage operation is planned, the patient lies on his stomach with a horseshoe pillow under his chest.

B. The skin incision starts at the level of the ninth dorsal segment, runs parallel with the lateral edge of the sacrospinalis muscle and then crosses the twelfth rib, swinging toward the umbilicus as in a lumbar sympathectomy The latissimus dorsi and the inferior posterior serratus muscles are cut; the edge of the sacrospinalis muscle and the lumbodorsal fascia are defined The entire twelfth rib, or if it is rudimentary, the eleventh rib, is resected or enucleated The lumbodorsal fascia is incised (Modified from Smithwick, R. H . Technic for Splanchnic Resection for Hypertension, Preliminary Report Surgery, 7 1, 1940)

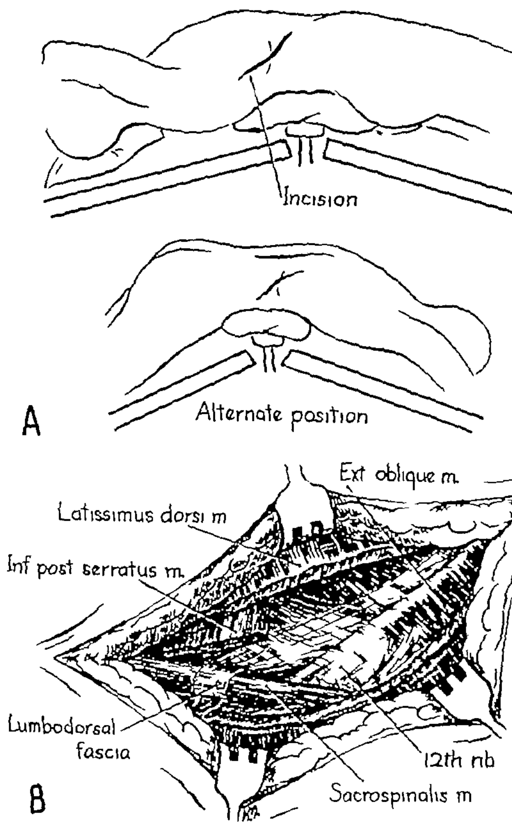

Incision
Alternate position
A
Ext oblique m
Latissimus dorsi m
Inf post serratus m
Lumbodorsal fascia
12th rib
Sacrospinalis m
B

Figure 371

Figure 372.

Both the pleura and peritoneum are reflected medially. The diaphragm extends like a curtain between them; the patient's head is to the right.

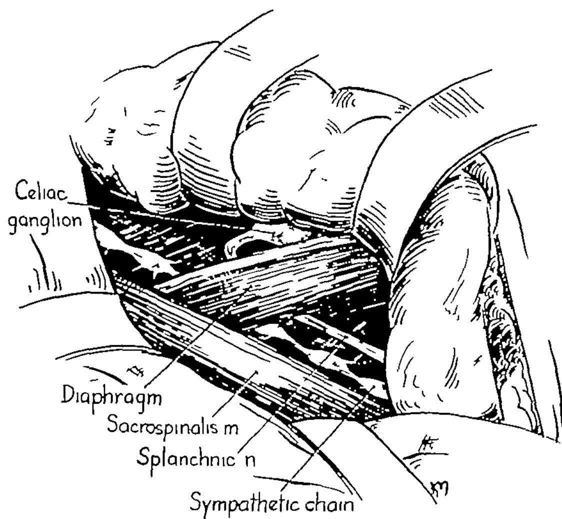
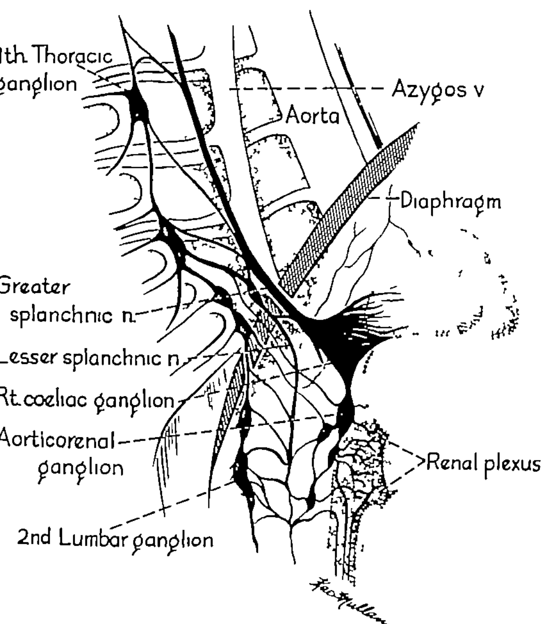

Celiac ganglion
Diaphragm
Sacrospinalis m
Splanchnic n
Sympathetic chain

Figure 372.

Figure 373.

The anatomy of the area as seen during adequate exposure is shown The greater splanchnic nerve is excised from the midthoracic level to its entrance into the celiac ganglion The sympathetic chain is removed from above the ninth thoracic to below the second lumbar ganglion. The diaphragm may be cut during this procedure or the medial crus simply incised in its tendinous portion and the structures pulled through The adrenals can be readily removed during the same procedure



th Thoracic ganglion
Azygos v
Aorta
Diaphragm
Greater splanchnic n.
Lesser splanchnic n.
Rt. coeliac ganglion
Aorticorenal ganglion
Renal plexus
2nd Lumbar ganglion

Figure 373

Figure 374.

A A generous pedicled flap is cut from the sacrospinalis muscle

B The renal capsule is stripped and the kidney scarified

C A small cortical wedge is removed for histologic section

D The muscle flap is swung into the renal defect for hemostasis and possible vascularization of the kidney

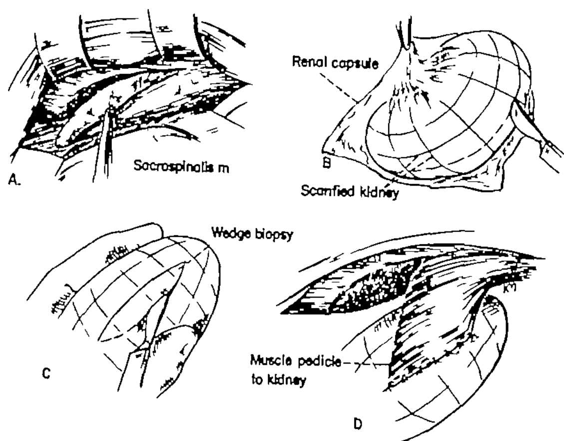
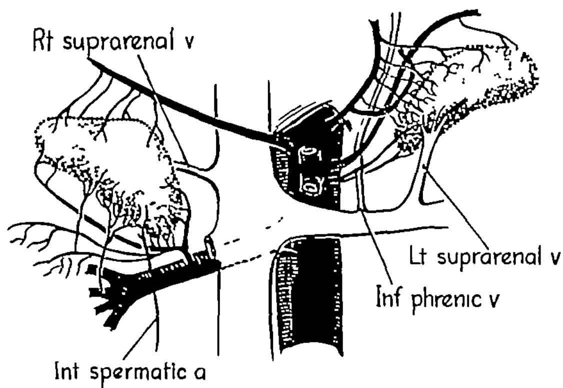

Renal capsule
Sacrospinalis m
A.
B
Scarified kidney
Wedge biopsy
C
Muscle pedicle
to kidney
D

Figure 374

ADRENALECTOMY

Figure 375. Adrenalectomy

Expose the adrenal gland as in a transdiaphragmatic splanchnicectomy by resecting the twelfth rib or by an incision coursing just below it If there is an obvious large tumor, an abdominal approach is preferable If at all possible, the gland should be isolated from the systemic circulation by doubly ligating and cutting its vascular connections Note that the pattern is different on the two sides A, and that the right suprarenal vein empties into the vena cava On the left, the inferior phrenic vein may join the suprarenal vein or it may empty directly into the renal vein, especially if the gland is hyperplastic. The various arterial patterns to the adrenal gland have been intensively studied by Anson and his co-workers [5] For exposure of the gland B, leave the renal capsule intact, and retract the kidney downward The yellowish, hard adrenal gland has the consistency of a rabbit's ear and can be readily distinguished from the surrounding fat.


Rt suprarenal v
Lt suprarenal v
Inf phrenic v
Int spermatic a
A

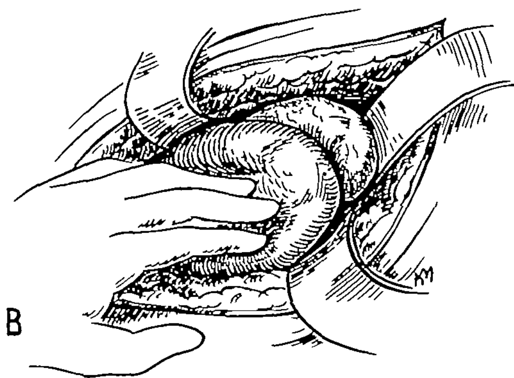

B

Figure 375

Figure 376.

Skin flaps for amputations (a) midthigh, (b) Callander, (c) lower leg, (d) midmetatarsal, and (e) toe

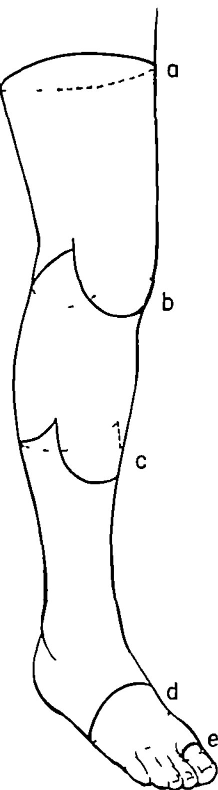


Figure 376.

These are no different from the classic methods of amputation as illustrated in any surgical text book. There are three important points to be observed however if amputation is to succeed at the lowest possible level in vascular disease.

(1) *Flaps* are minimized. In metatarsal amputations a plantar flap is unavoidable but is fortunately fairly well vascularized. No dorsal flap is ever used. Necrosis of the flap occurs here more readily. Lower leg and thigh amputations need only enough flaps to avoid dog ears and the three phase guillotine principle is utilized. Cutting the skin with the deep fascia, muscle and bone at three progressively higher levels is important. Figure 376 shows the skin flaps for midthigh, Callander, lower leg, midmetatarsal and toe amputations. A Syme operation may occasionally succeed but, except in Buerger's disease or frostbite, it carries considerable risk.

(2) *Muscle and fascia* are not sutured over the bone; they are cut short and allowed to retract. This causes considerable raising of the eyebrows among orthopedic surgeons. However, sloughing muscle and fascia delays healing in ischemic extremities. Plasma and infected lymph can drain well through large drainage tubes placed through stab wounds posterior to the skin suture. This prevents bulky fibrinous exudates leading to stump fibrosis.

(3) The *skin* is closed with *no tension*; it should fall together with only a few loose stainless-steel wire or silkworm gut sutures approximating it. If the skin fits too well at the completion of the amputation, there will be a *tight stump* a few months later. In the presence of acute infection, the skin should be left wide open and packed with water-soluble Furacin; a delayed secondary suture is done five to seven days later under an antibiotic umbrella.

These three principles apply to all forms of amputation. It hardly needs to be emphasized that a tourniquet is never used.

Figure 377. Midmetatarsal Amputation

This amputation is very useful in thromboangitis, in diabetes with neurotrophic lesions and in frostbite It is seldom applicable in arteriosclerosis

A A large plantar flap is cut clear to the metatarsal bones, but the dorsal incision is straight

B All five toes are sawed with a Gigli saw.

C The suture line is dorsal, weight bearing is adequate with a foam rubber pad glued into the shoe in the place of the toes.

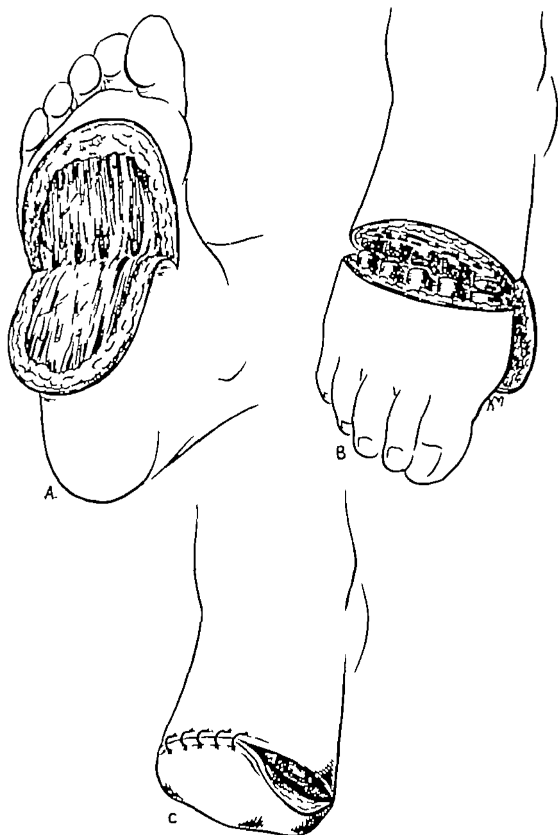


Figure 377

Figure 378. Amputation of the Lower Leg

A The patient is in a prone position A circular skin incision is made 6 to 7 inches below the tibial tubercle.

B The skin with the muscle fascia is dissected up The tibia and fibula are stripped from the periosteum, the tibia is divided 3 to 4 inches above the skin incision, the fibula 1 inch higher All muscles are cut short to avoid a bulky stump. Veins and arteries are tied with fine cotton Nerves are tied, not retracted and not injected with alcohol

C The skin is allowed to fall in place It is loosely united, with a drain lead out through the posterior flap and secured with a long ligature so that it may be removed in 48 hours without disturbing the dressing (Modified from Smith, B C Amputation Through the Lower Third of the Leg for Diabetic and Arteriosclerotic Gangrene Arch Surg, *27* 267, 1933.)

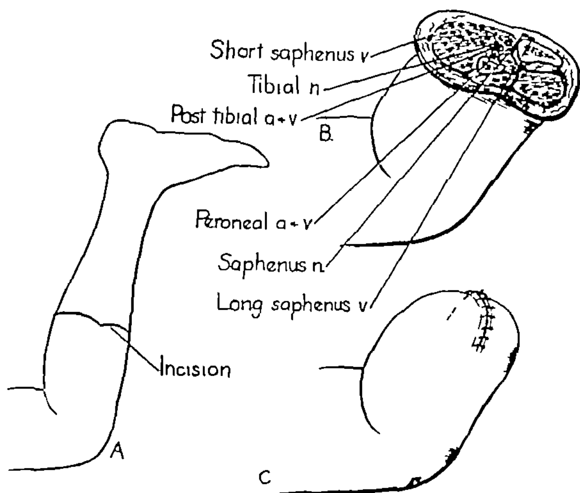

Short saphenus v
Tibial n
Post tibial a + v
B.
Peroneal a + v
Saphenus n
Long saphenus v
Incision
A
C

Figure 378

Figure 379. Tenoplastic Transcondylar Amputation of Callander

A Flaps are unavoidable here, but they are usually vascularized

B. The medial hamstrings are cut close to their insertion at their tendinous portions The insertion of the adductor muscle is not shown.

C. The lateral aspect of the thigh is seen, and the cut tendons of the tensor fasciae latae and biceps muscles are indicated The edge of the quadriceps femoris muscle is visible in the upper right corner The index finger has picked up the nerves and blood vessels from the posterior surface of the femur.

D The anterior flap has been turned up and the knee joint is exposed The patella is to be excised, preserving the patellar ligament The cruciate ligaments will be severed

E. The femur has been divided through its condyles. No sutures were placed in the cut tendons, which are allowed to retract The bed of the patella fits the sawed surface of the femur. The skin flaps fall together and are loosely united with skin clips. The drain is placed posteriorly.

This is an excellent amputation for vascular disease, but is disliked by limb manufacturers and prosthesis clinics because the stump has a wide condylar flare and is not conical It can be fitted well, however, with some effort on the part of the limb maker [6] (Callander, C L · New Amputation in in the Lower Third of the Thigh J A M.A , *105*.1746, 1935)

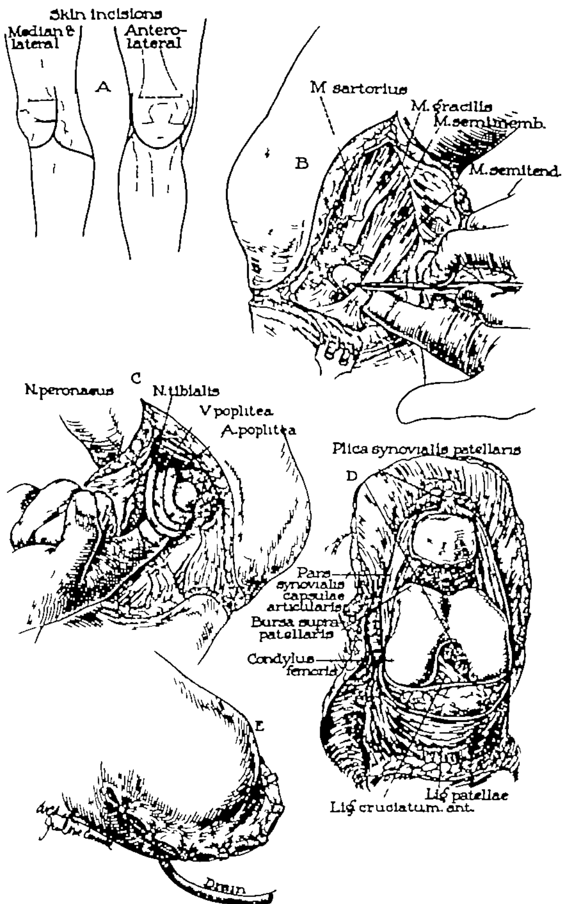
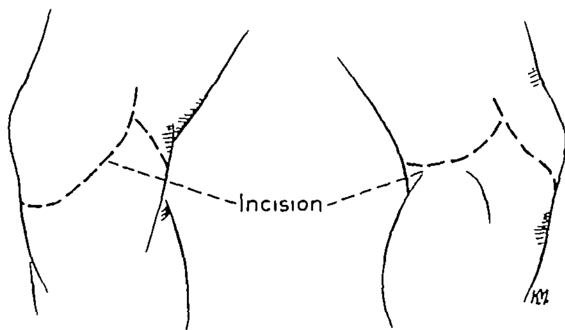

Skin incisions
Median & lateral
Antero-lateral
A
M sartorius
M. gracilis
M. semimemb.
B
M. semitend.
C
N. peronaeus
N. tibialis
V poplitea
A. poplitea
Plica synovialis patellaris
D
Pars synovialis capsulae articularis
Bursa suprapatellaris
Condylus femoris
Lig. patellae
Lig. cruciatum. ant.
E
Drain

Figure 379

Figure 380. Supracondylar Amputation in the Lower Third of the Thigh

The skin incision and surgical anatomy are identical with those shown in figure 379. At the supracondylar level, however, large muscle bellies have to be cut, more ligatures are needed and more trauma is produced In the cross section, these muscle masses are obvious. The only possible reason for using this amputation is that the stump can be fitted more easily and is suitable for the use of a suction limb.


Incision
KM

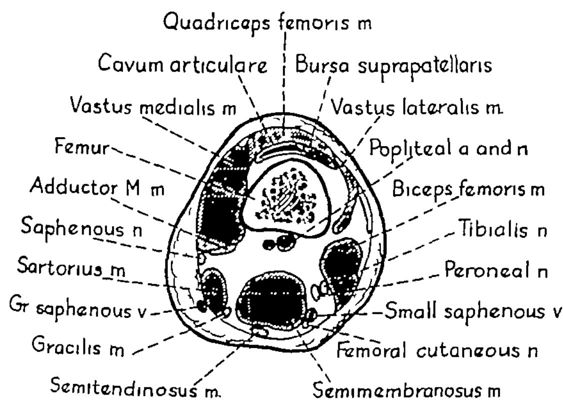

Quadriceps femoris m
Cavum articulare
Bursa suprapatellaris
Vastus medialis m
Vastus lateralis m.
Femur
Popliteal a and n
Adductor M m
Biceps femoris m
Saphenous n
Tibialis n
Sartorius m
Peroneal n
Gr saphenous v
Small saphenous v
Gracilis m
Femoral cutaneous n
Semitendinosus m.
Semimembranosus m

Figure 380

Figure 381.

A The posterior flap exposes the contents of the popliteal fossa The horseshoe incision, with a split in the gastrocnemius seam, is an excellent exposure for the popliteal bifurcation

B. The anterior aspect shows the patella with the quadriceps tendon

C and D show the closure of skin with the site of drainage

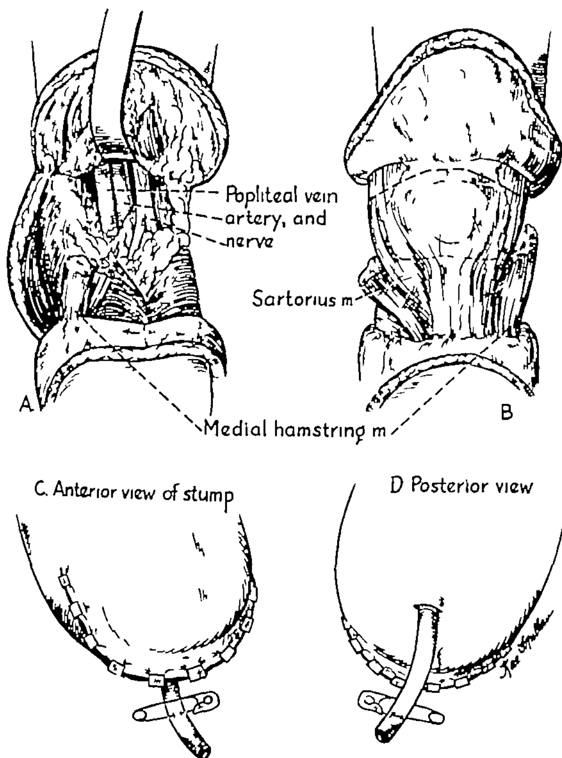

Popliteal vein
artery, and
nerve
Sartorius m
A
B
Medial hamstring m
C. Anterior view of stump
D Posterior view

Figure 381

Figure 382. *Rehabilitation of the Patient after Amputation*

The philosophy of total medical care is particularly important for the amputee. His personal, social and vocational problems should be investigated and the surgeon must be sufficiently aware of them to direct the patient properly Prosthesis clinics and institutes of rehabilitation do excellent work in instructing the patient to use walkers, peglegs or a definitive limb [7] This 83 year old carpenter, who lost his leg above the knee, was walking on the fifth day, had a temporary leg on the twenty-first day and used an artificial limb for three years He needed little outside help.

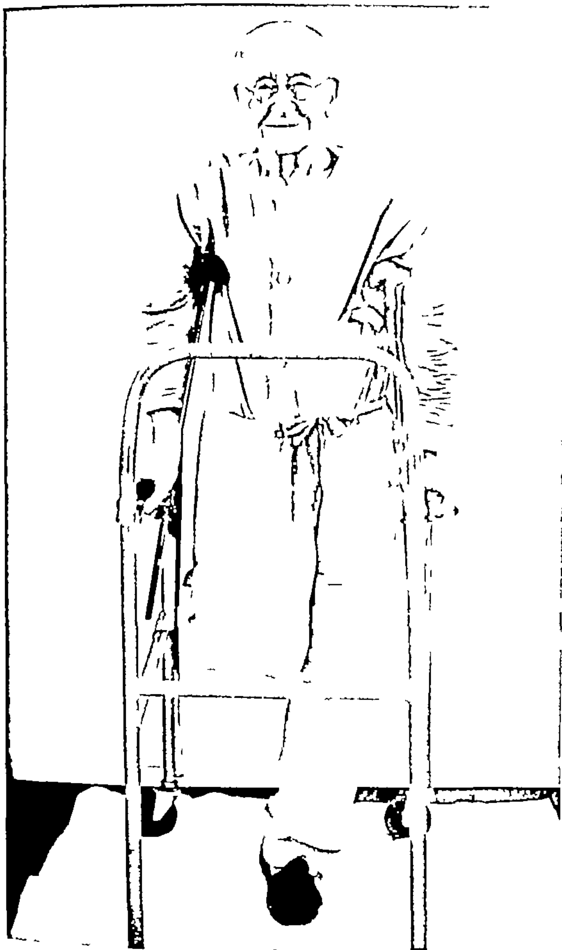


Figure 382.

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